GIT

Version 5.2

Corrected, Updated, Lighter

PLAB 1 Keys is for PLAB-1 and UKMLA-AKT (Based on the New MLA Content-Map)

With the Most Recent Recalls and the UK Guidelines

ATTENTION: This file will be updated online on our website frequently!

(example: Version 2.1 is more recent than Version 2, and so on)

Key 1	**Celiac Disease** ◘ Autoimmune, Malabsorption disease, results due to sensitivity to **Gluten** (which is a protein). ◘ Eating gluten diet (e.g., **Rye**, **Wheat**, **Barley**) → **Villous atrophy** of the GIT → Malabsorption → Iron deficiency Anemia, Folic Acid and Vit. B12 Deficiency, malabsorption of fat.

Manifestations

- Chronic or Intermittent **Diarrhea**.
- **Steatorrhea** (fatty stools due to malabsorption of fat).
- **Stinking**, bad-smell, **stools**
- **Abdominal discomfort, Bloating, Nausea and Vomiting.**
- **Wight Loss. √**
- **Iron deficiency anemia** (the most common), followed by **Folate deficiency** then **Vit B12 deficiency**.
- Manifestations of anemia e.g., **Fatigue**.

Complications → *Osteoporosis* / *T-cell lymphoma* (rare).

- **Association not to be forgotten** → **Dermatitis Herpetiformis**.

Diagnosis

- Positive **TTG** and **IgA**. (First Line)

  (TTG= **Tissue TransGlutaminase Antibodies**)
- Positive Endomysial Antibodies.

Important: If the serum tissue transglutaminase antibodies are negative but the clinical presentation is still suggestive of celiac disease (eg, diarrhea,

intermittent abdominal ache especially after consuming gluten diet eg, wheat) and in the presence of serum IgA deficiency)

→ Perform serum tissue transglutaminase antibodies using an IgG-based essay. After that, arrange for jejunal/ duodenal biopsy to confirm the diagnosis.

If TTG is positive, we need to confirm the diagnosis of Celiac disease by Biopsy → **Jejunal or Duodenal Biopsy.** It will show:

- **Villous Atrophy.**
- **Crypt hyperplasia**.
- ↑ inter-epithelial **lymphocytes**.

Important: for the biopsy to be accurate, the patient should re-introduce the gluten in his diet for 6 weeks before the biopsy.

Treatment → **Gluten-free diet**.

Example scenario:

33 Y/O male, Non-smoker.

Presents with recurrent and chronic diarrhea for 6 months.

His clothing appears to be ill-fitting (indicative of weight loss).

Hb = 11 ▌MCV = 105 (high)

	o The most likely Diagnosis → **Celiac Disease**. o Endoscopy + Duodenal Biopsy will show → **Villous Atrophy**. **Asked Before:** **Q) Why there is malabsorption in celiac disease patients (what is the pathophysiological reason for steatorrhea, anemia in celiac disease)?** → Villous atrophy in the small intestine (ie, decreased surface area for absorption).
Key 2	**Crohn's Disease VS Ulcerative Colitis** **◙ Points towards Crohn's disease** o It can affect any part of the GIT (from mouth to anus). o Endoscopy → **Skip lesions**, **Transmural** (deep Ulcers), **Cobblestone** appearance o Histology → **Granuloma**, **↑ Goblet cells**. o Examination → Abdominal Pain or Mass on the **RIGHT** iliac fossa. o **Diarrhea** "Usually **Non-bloody but can be bloody**". o **Weight loss** is more common. o **Fistulae, perianal fistulas, anal fissures.**

◙ Points towards Ulcerative Colitis

- Affects the mucous membrane starting from rectum.
- Barium enema → **Loss of haustration**, **drain pipe** appearance.
- Histology → **Crypt Abscesses**, **(↓) Goblet Cells**.
- Abdominal pain on **LEFT** lower quadrant.
- **Bloody Diarrhea** is more common.
- **Primary Sclerosing Cholangitis** is more common.

Aphthous oral ulcers can be seen in both CD and UC, however, slightly more common in **CD**.

Notes:

√ Smoking **increases** the risk of **CD**.

√ Smoking **decreases** the risk of **UC** "protective".

Notes:

- **Crohn's disease → colonoscopy → Cobble stone appearance, Deep ulcers, Skip lesions.**
- **Crohn's disease → Small Bowel Enema → Kantor's string sign, thorn ulcers and fistulae.**
- **Ulcerative colitis → Barium enema → Loss of haustral markings.**

◙ **The most appropriate investigation:**

• It is a usual practice to **initially** perform stool culture and microscopy for any one with chronic diarrhea.

• However, the **most appropriate investigation** for **Crohn's** disease (eg, chronic diarrhea, anal fissures/ fistulae, abdominal pain) is → Colonoscopy.

During colonoscopy, the doctor will assess the gross features of Crohn's and will take biopsies of the affected colonic segments to look for microscopic evidence of Crohn's disease: Skip lesions, Transmural (deep Ulcers), Cobblestone appearance, Granuloma, ↑ Goblet cells.

◙ **Treatment of Inflammatory Bowel Disease in Short:**

• **Crohn's Disease** → Oral **Prednisolone** (**1st line for remission**). Or **budesonide**

If both are NOT given in the options, pick **Mesalazine** **(as it is the 2nd line)**

Mnemonic: **C**rohn's → **C**orticosteroids (prednisolone) 1st line.

• **Ulcerative Colitis** → **5-ASA** (**Mesalazine**). (**1st line to induce remission**)

• **severe UC exacerbation (Toxic Megacolon)** → Pick **IV Hydrocortisone**.

o Mnemonic: **C**rohn's → **C**orticosteroids (prednisolone) 1st line.

Key 3

Barret's Oesophagus

Under the prolonged hydrochloric acid reflux to the oesophagus (e.g. in those having GERD) → the **lower oesophagus** undergoes "**Metaplasia**" which means that the epithelium lining the mucosa of the lower oesophagus will change from **Squamous** to **Columnar** epithelium.

[**Squamous** epithelium turns to **Columnar** epithelium **with goblet cells**]

S → **C**

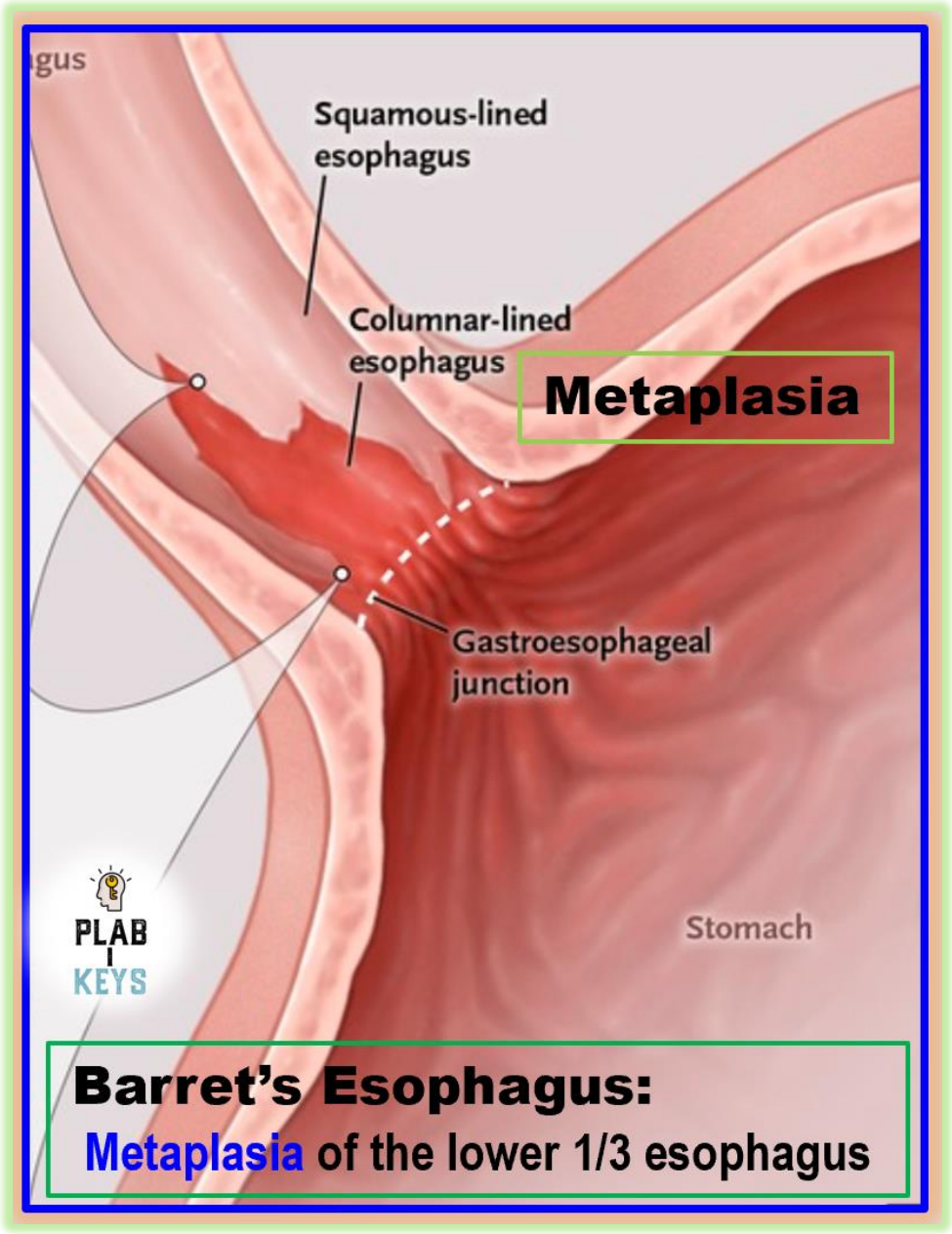
Shampoo for **C**hildren

Squamous → **C**olumnar

Therefore, the change that is expected to be seen on the histology of the lower third of oesophagus in patients with Barret's oesophagus is:

→ **Columnar Metaplasia**.

This is a precancerous condition as it can develop into oesophageal **Adenocarcinoma** of the lower 1/3 of the oesophagus.



N.B.

Achalasia → **SCC** of the **upper 2/3** of the oesophagus.

Barret's → **Adenocarcinoma** of the **lower 1/3** of the oesophagus

	(Adenocarcinoma of the oesophagus is Common in **GERD** and **Barret's oesophagus)**.
Key 4	**Achalasia** • **Inability to relax the lower oesophageal sphincter (LOS)** due to the idiopathic loss of the normal neural structure. **i.e.** (↑ Lower Oesophageal Resting **Pressure**). • Presents with **Progressive Dysphagia to both solids and liquids**. • The word "**Regurgitation**" should draw your attention towards either **Achalasia** OR **Pharyngeal pouch**. o In Pharyngeal pouch, however, there are other specific features such as (Halitosis) = Bad breath smell, (stale food or fluid), (gurgling sound in the chest when drinking), (Sensation of a lump in the throat), neck bulge. **Important Note:** If the dysphagia is progressive and associated with significant weight loss in an old individual, suspect oesophageal carcinoma even if there is gurgling sounds on drinking (it also occurs in cancer).

- Regurgitation can lead to → Aspiration Pneumonia

  → productive cough and fever.

- Remember, Achalasia has no relation to tobacco or alcohol while Oesophageal cancer has.

- There might be weight loss, chest pain in achalasia.

- **Investigations**

  - **X-ray** → **Megaoesophagus** = Large "Dilated" Oesophagus.
  - **Barium meal** → **Bird's Beak** Appearance of the distal end of the oesophagus.

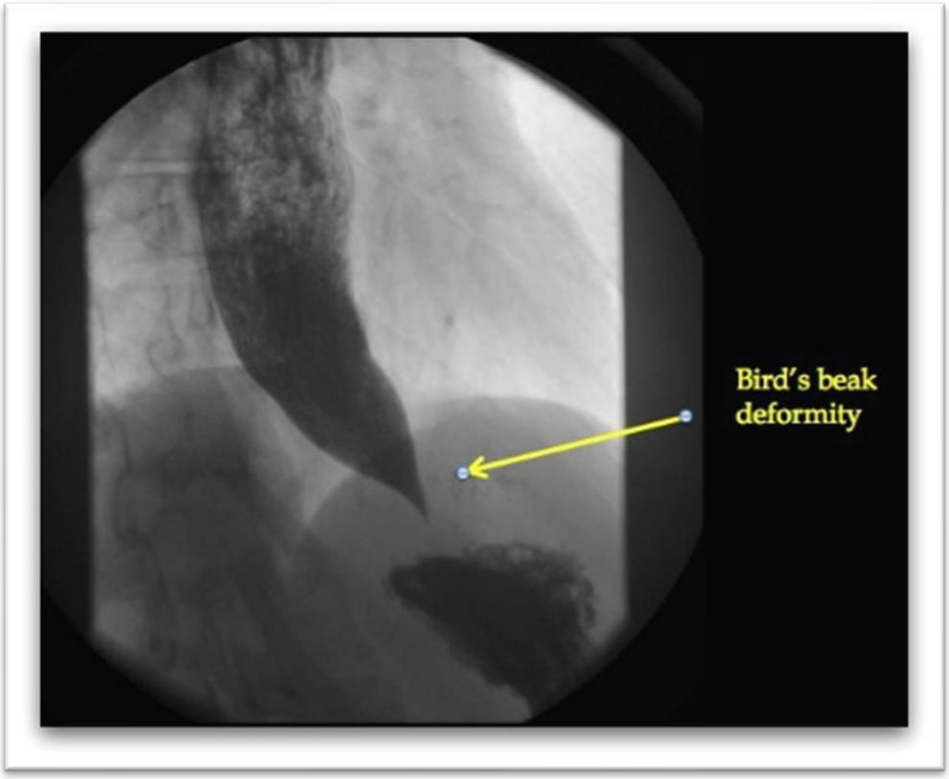
    (**Dilated Oesophagus that tapers -Narrows- Towards the distal end of the oesophagus**)

  - The most accurate

    → **Oesophageal Manometry**

    → **Increased lower oesophageal resting pressure**.

- **Treatment** → **Dilatation of the lower oesophageal sphincter**.



Example Scenario:

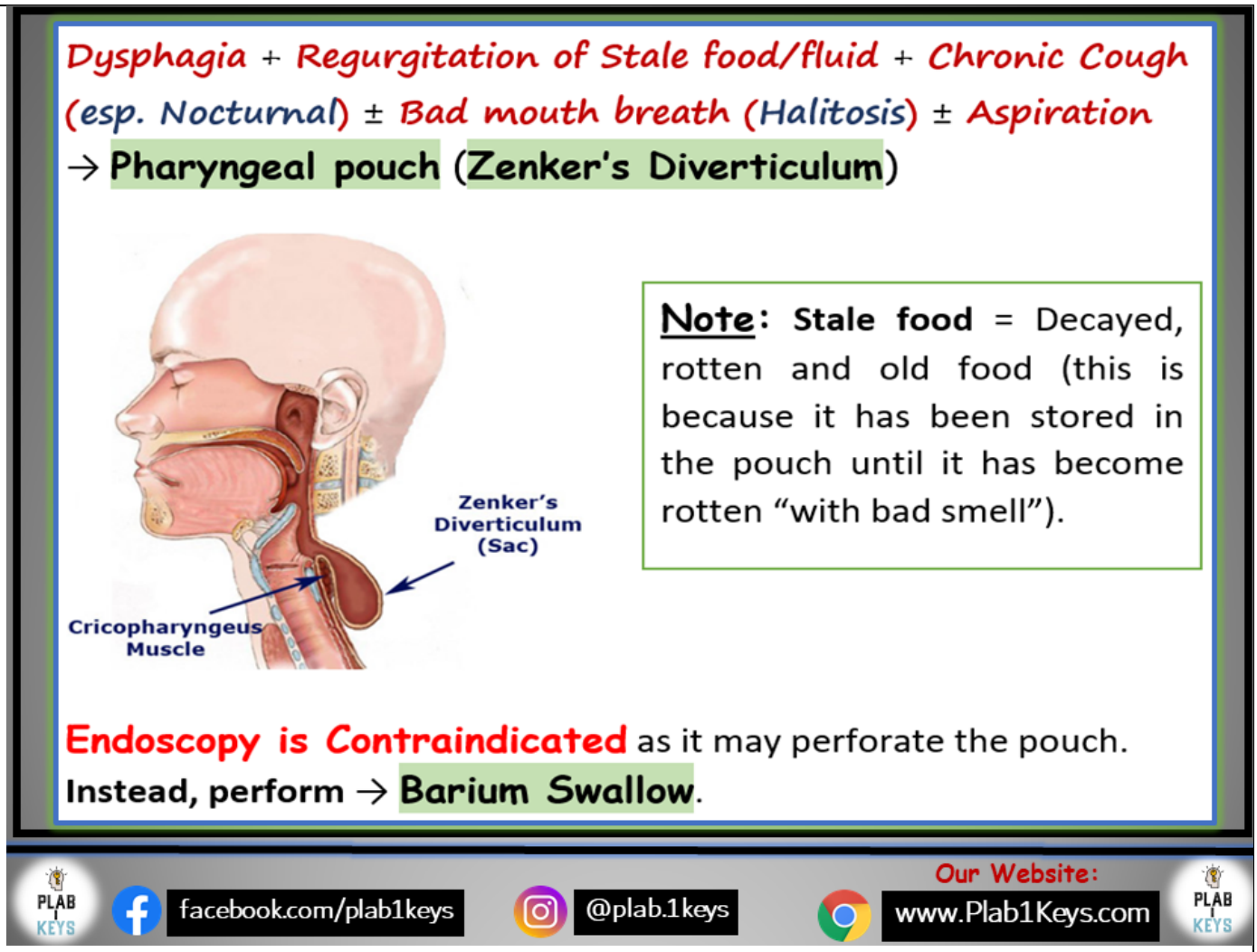
45 Y/O woman presents with productive cough and moderate fever. She also complains of central chest pain, **Regurgitation** of undigested food and Dysphagia to both solids and fluids.

Chest X-ray shows → **Megaesophagus**.

The likely Diagnosis → **Achalasia**

	Productive cough and moderate fever → Aspiration pneumonia due to the regurgitation.
Key 5	**In the previous example, Why Not Oesophageal Cancer?** **Risk Factors That would raise our suspicion to oesophageal cancer:** *Old age* ± *weight loss* ± Hx of *smoking, Alcohol* ± *Anemia* ± ***Increasing*** *"Progressive" dysphagia to solids then to Fluids* ± *Hx of Barret's Oesophagus* ± *Hx of GERD* • **The diagnostic Investigation of oesoph. Cancer →** **Endoscopy + Biopsy**. **Notes on Oesophageal Carcinoma** o The most common type → **Adenocarcinoma**. o Smoking → Associated more with **SCC**. o Barret's Oesophagus → a precursor of **Adenocarcinoma**. o Achalasia → chronic inflammation → more risk for **SCC**. o Upper 2/3 of the oesophagus → **SCC**. o Lower 1/3 of the oesophagus → **Adenocarcinoma**.

Key 6



Also, gurgling sounds in the chest when drinking is an important sign for **pharyngeal pouch**.

Important Note:

If the dysphagia is progressive and associated with significant weight loss in an old individual, suspect oesophageal carcinoma even if there is gurgling sounds on drinking (it also occurs in cancer).

Key 7

Villous Adenoma

- Frequent episodes of **Excessive Watery** Diarrhea + Large amounts of **mucous**.
- It is one of the causes of **Metabolic Acidosis** and **Hypokalaemia**. √
- Endoscopy → Cauliflower like mass.

Remember that, in Villous Adenoma, it secretes mucous which is rich in protein and potassium → thus, hypoproteinaemia and Hypokalemia.

◙ Important Causes of HypOkalemia

√ **Loop diuretics (e.g., furosemide).**

√ **Thiazide**-like **diuretics** (e.g., **Bendroflumethiazide**).

√ **Vomiting** and **Diarrhea**.

√ **Villous Adenoma**.

√ Renal tubular failure.

√ **Cushing's syndrome**.

√ **Conn's** (1ry Hyperaldosteronism): hypokalemia with hypertension.

√ Bartter's syndrome: (a child: failure to thrive, hypokalemia with normotension, polyuria, polydipsia, weakness, usually autosomal recessive).

	◙ **Important Causes of HypeRkalemia** **√ ACE inhibitors (e.g. enalapril, captopril).** **√ Potassium sparing diuretics (e.g. Spironolactone**). **√ CKD (Chronic Kidney Disease)**. **√ Addison's disease** (Hyperkalemia, postural hypotension, hyponatremia, hypoglycemia)
Key 8	**Vitamin B12 Deficiency** "***updated***" **Megaloblastic Anemia** = low Hb, **high** MCV → Either **vitamin B12** deficiency or **Folic acid** deficiency or both. Vitamin B12 = Cobalamin. ◙ **Causes of Vitamin B12 deficiency** o **Pernicious Anemia** (The most common cause). Pernicious Anemia → Autoimmune Gastric Atrophy → ***Loss of intrinsic factors*** that are required for Vit B12 absorption. Usually associated with other autoimmune diseases eg, hypothyroidism. o **Total Gastrectomy** (Impaired Vit B12 Absorption).

- **Ileal Resection**. (Malabsorption: the majority of vit B12 is absorbed in the terminal ileum)
- **Crohn's Disease**.
- **Chronic Pancreatitis** (malabsorption).
- **Celiac Disease** (malabsorption).
- **Dietary** (Vegans). Remember that Vit B12 is present in **meat**, **fish** and **dairy products** but not in the vegetables. Folic acid is in green vegetables.

Thus, vegans often develop vitamin B12 deficiency.

And, those who do not eat vegetables often develop Folate deficiency.

Features of Vitamin B12 deficiency

- **Peripheral paraesthesia**.
- **Impaired position and vibration (proprioception) sense**.
- **Dementia** → *loss of memory* + *difficulties with thinking*.
- If untreated → permanent **Ataxia**.

Lab Results of Vitamin B12 deficiency

- **↑ MCV** (usually > 110) + ↓ Hb: Macrocytic Anemia.
- **Hypersegmented Neutrophils on a blood smear**.
- ↑ Homocysteine.

These Lab results are also present in Folic Acid deficiency.

So, How to Differentiate?

By the History:

- **Vegans** (who do not eat meat, fish, dairy products) → Vit B12 deficiency.
- If the patient **does not eat vegetables** → Folic Acid deficiency.
- Gastric or ileal resection → Pernicious Anemia → Vit B12 Deficiency.

◙ **Treatment of Vitamin B12 Deficiency**

→ **IM Hydroxocobalamin (ie, Vitamin B12 IM injections)**.

Quick Scenario (1):

A man has **low serum folate** → Encourage him to eat **leafy green vegetables**.

Quick Scenario (2):

A man has **low serum folate and low vitamin B12**

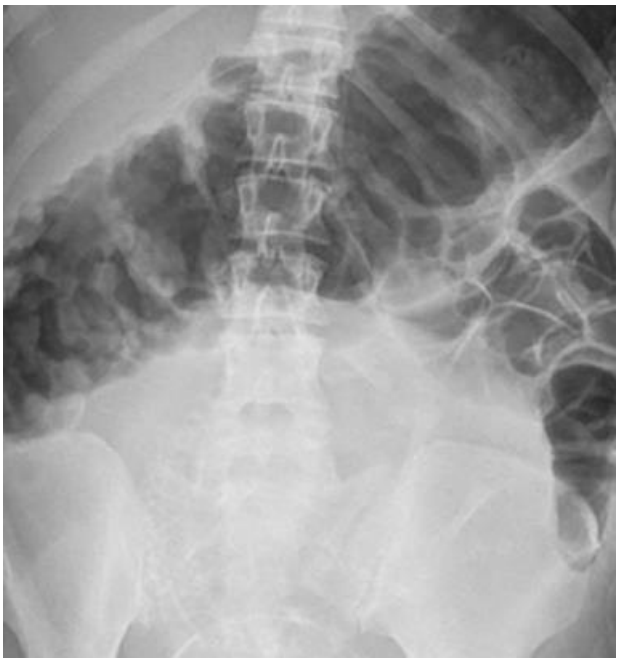
→ **Treat vitamin B12 deficiency first (eg, give vitamin B12 injections)**.

(Mnemonic: **B** before **F** → treat vit **B**12 deficiency before **F**olate deficiency)

Quick Scenario (3):

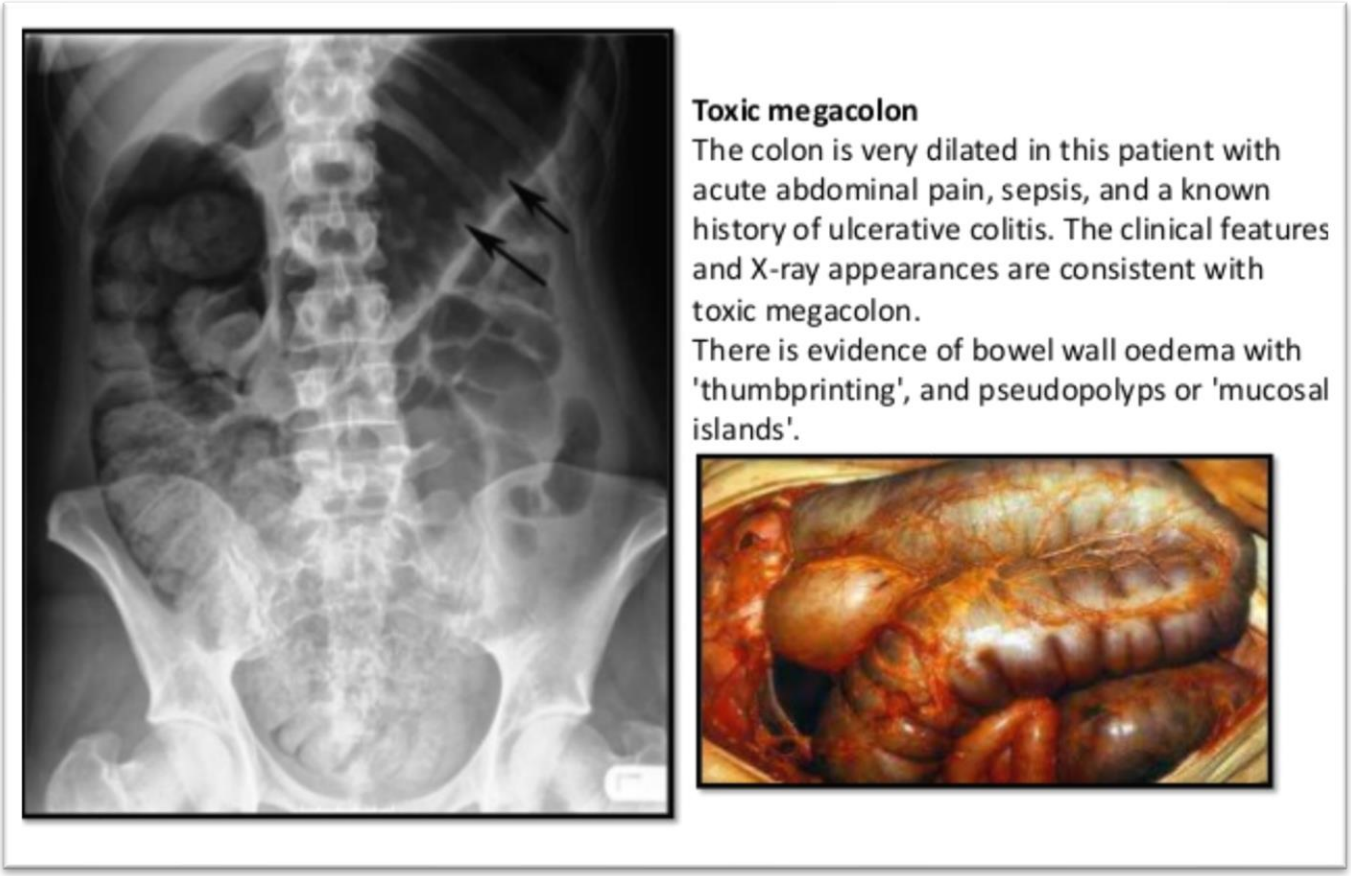
	A young man presents with several attacks of pancreatitis. He has peripheral paraesthesia, loss of proprioception in his legs, loss of memory and difficulties with thinking. **Dx→ Vitamin B12 Deficiency.** **Rx → Hydroxocobalamin (ie, vitamin B12 IM injections).**
Key 9	**A patient with a Known Ulcerative Colitis presents with high frequency diarrhea (8 times a day) + Visible Blood. The patient is pale and tachycardic (100 bpm) and with temperature of 38. The abdomen is tender with no palpable masses.** **Dx** → **Acute flare of UC**. (**Acute exacerbation of UC**) **Ix** → **Abdominal X-ray** → Toxic Megacolon (Mucosal edema and colon distension especially the transverse colon). Rx → **IV Hydrocortisone**. **When to suspect severe colitis (Acute Exacerbation of UC)?** The rule of **6**, **30**, **90** • > **6** bowel movements a day + Visible blood in large amounts. • ESR > **30** (also WBC may be elevated). • HR > **90** "mildly tachycardic"

- Temperature > 37.8 “mild feverish”
- Anemia (low Hb): (Pallor, Fatigue …etc).



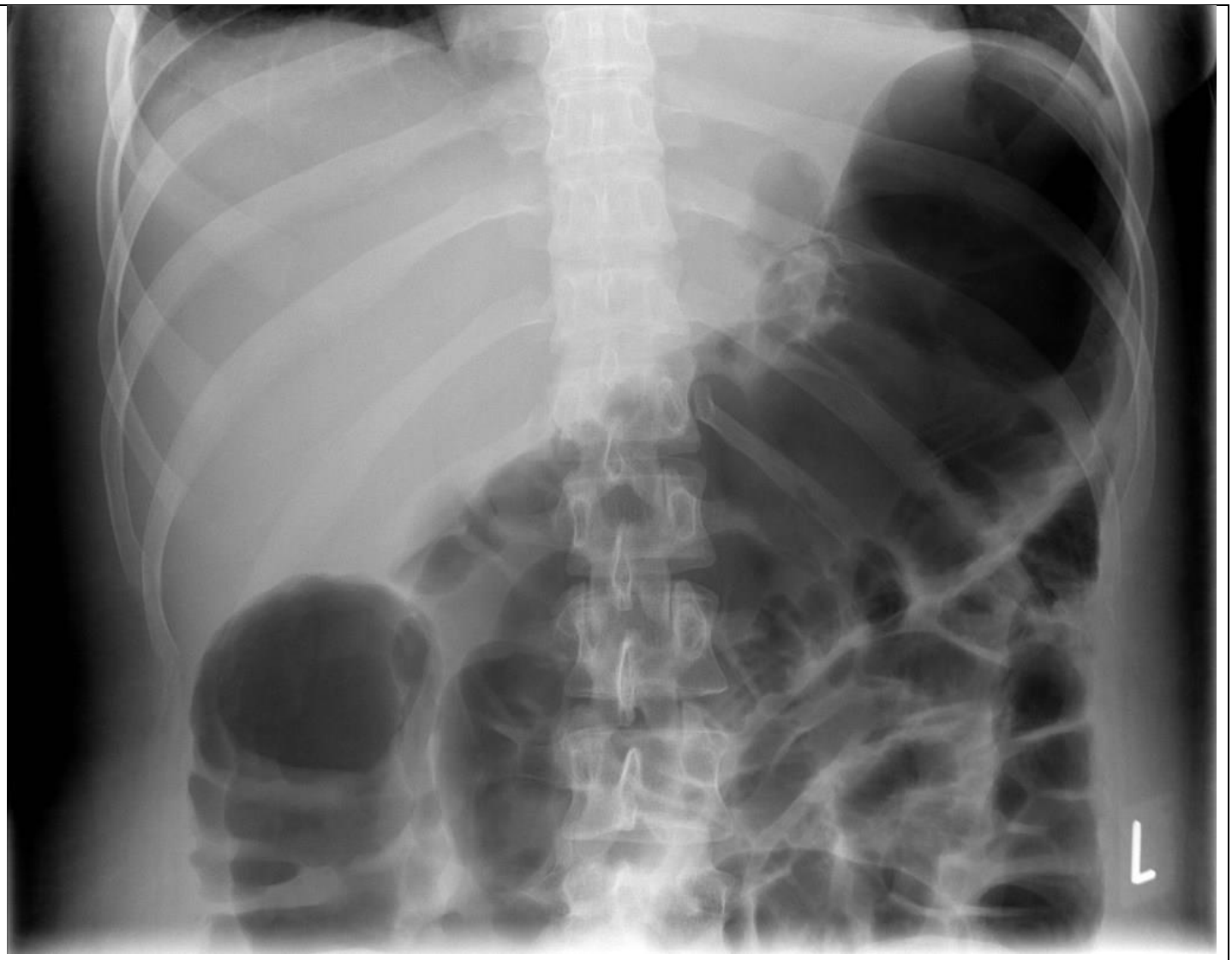
Toxic Megacolon on Abdominal X-ray

→ ***Dilated colon with thumb printing (Mural Edema) especially in the transverse colon.***



Important: sometimes, if a patient with toxic megacolon is septic, shortness of breath can develop.

In a recent exam, the features of toxic megacolon along with a picture of x-ray were given and was asked about the diagnosis.



Toxic megacolon in a patient with a background of ulcerative colitis who presented with shortness of breath, bloody diarrhea, fever and abdominal pain.

Key 10

Abnormal LFTs (Liver Function Tests)

\+ **2ry Amenorrhea**

\+ Young-middle age **Female**

→ Think of: **Autoimmune Hepatitis**

- ALT, AST, Bilirubin: (↑)
- Alkaline Phosphatase (ALP): **Normal** or **Mildly** (↑).
- The presence of another **associated autoimmune disease** would make the diagnosis of autoimmune hepatitis much easier.

  Examples: ***Vitiligo***, ***Addison's***, autoimmune ***thyroid*** disease, ***DM*** 1.
- It tends to progress to *liver cirrhosis*.

Alcoholic Liver Disease.

♦ Hx of heavy alcohol consumption.

♦ Signs of liver disease/ cirrhosis: Ascites, Hematemesis, Jaundice, Hepatomegaly, Spider naevi.

♦ In Alcoholic liver disease:

Both AST and ALT are elevated; however, AST is more elevated than ALT

→ **↑ AST:ALT ratio** (e.g. AST:150, ALT: 70).

♦ In Alcoholic liver disease:

Gamma Glutamyl Transferase **(GGT) is also increased**.

♦ Management?

◙ Stop Alcohol

◙ Consider Liver transplant 6 months after alcohol abstinence in late cases.

Key 11

HELLP Syndrome

VS

Acute Fatty Liver of Pregnancy (AFLP)

- **HELLP** Syndrome → **H**emolysis, **E**levated **L**iver enzymes, **L**ow **P**latelets.
- AFLP → **ELLP** (without **H**emolysis) + (↓) Glucose ± (↑) Ammonia

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- In HELLP Syndrome, there is **Haemolysis** (Low Hb would be given).

	• In AFLP, in addition to ELLP (Elevated Liver enzymes, Low Platelets), there is Low serum glucose ± High serum Ammonia. Also, AFLP has **vomiting** ± Disseminated Intravascular Coagulopathy (**DIC**) "Prolonged PT, PTT". **Acute Fatty Liver of Pregnancy** o **RFs** → Pre-eclampsia, First pregnancy, Multiple Pregnancies. o **Presentation** → Nausea, Vomiting, Abdominal Pain, Fever, Headache, Pruritus, Jaundice. o Occurs late in pregnancy (Typically **after 30 weeks of gestation**) and may occur immediately after delivery. o It is rare but life-threatening (***severe hypoglycemia and abnormal clotting factors → coma → death***). o **Diagnostic** → Liver Biopsy.
Key 12	**Intermittent diarrhea + abdominal bloating + Fatigue** Think→ **Celiac Disease**.
Key 13	**Acute Cholecystitis** • **Presentation** o **Acute severe right upper quadrant pain or epigastric pain that may radiate to the back or to the right flank or to right shoulder.**

- **May be precipitated by meals.**
- **± Nausea, Vomiting, Fever.**
- Jaundice can develop if there are stones in the common bile duct (**CBD**) "*Choledocholithiasis*". However, in Acute cholecystitis due to stones, the stones are usually blocking the "**Cystic Duct**" = the neck of the gallbladder; therefore, the jaundice is often absent or mild to notice.
- **Positive Murphy's sign → pain and arrest of inspiration when pressing the right costal margin at the midclavicular line**.

  Remember, this sign is sensitive but NOT specific to acute cholecystitis.
- ± Deranged LFTs.

- **Inflammatory element: fever, ↑ WBCs.**
- Note, ↑ ALP (Alk. Phosphatase) → indicates a sort of biliary obstruction with cholestasis.

  This might occur with acute cholecystitis.
- > 90% of Acute cholecystitis cases are due to blockage of cystic duct by a gallstone.

- **Risk Factors**

√ Note, acute cholecystitis is quite common during **pregnancy**.

√ Other risk factors include: **Female** gender, **Obesity**, **Weight Loss**.

	Remember: **5-F** Syndrome: [**f**air, **f**at, **f**emale, **f**ertile and over **f**orty]. However, this does not mean that acute cholecystitis does not occur in men, thin, under forty years old! • **Investigation** ↑ WBCs **U/S Abdomen** → Thick walled and Shrunken Gallbladder. • **Treatment** √ Nothing per mouth / IV Analgesics / IV fluids / IV Antibiotics. √ NICE now recommend **early laparoscopic cholecystectomy, within 1 week of diagnosis**. Previously, surgery was delayed for several weeks until the inflammation has subsided. ***Note***: The main difference from Biliary colic is that acute cholecystitis has an **inflammatory element** (**↑ WBCs**, **fever**, **Local peritonism** "Tenderness")
Key 14	• Incidental Finding of **Gallstones** in an **Asymptomatic** patient during CT or U/S → **Reassurance** (No intervention is needed)

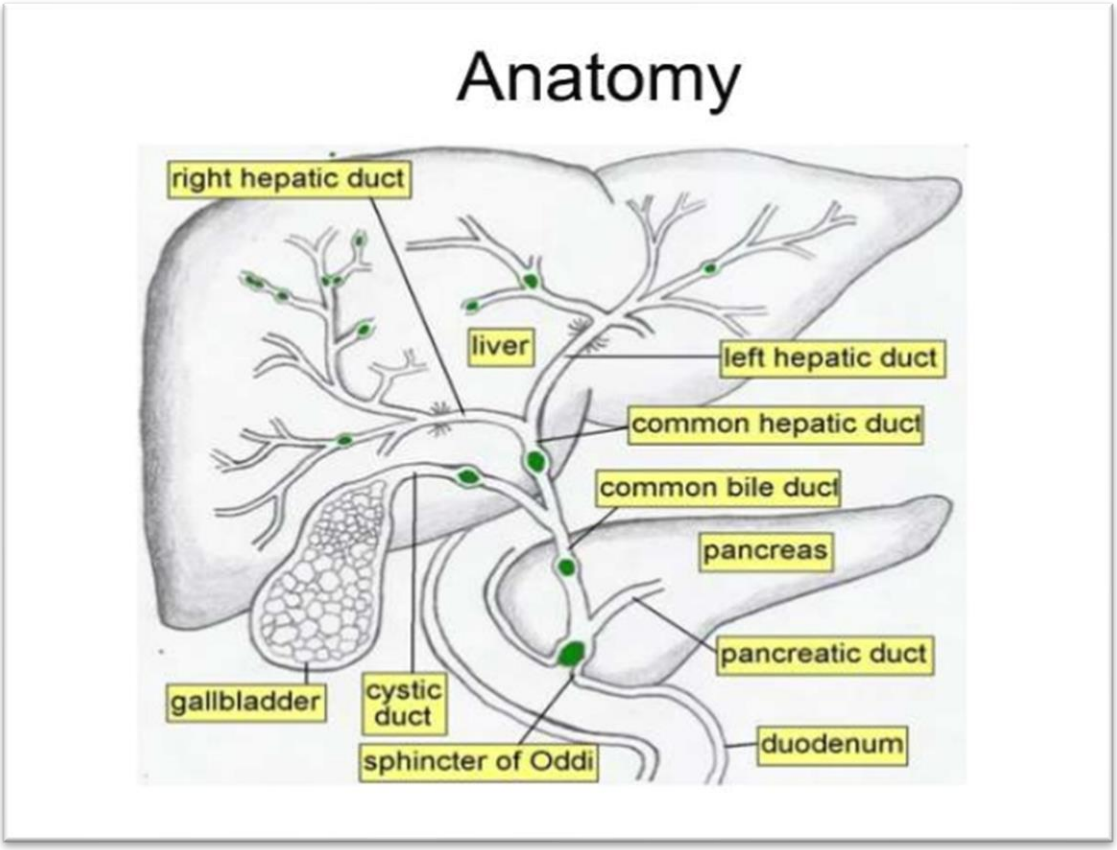
- Note: If the **U/S** shows stones in the **Common Bile Duct** (CBD) "Choledocholithiasis" instead of the Gallbladder even if in an **Asymptomatic patient**

  → **ERCP** or **Laparoscopic Cholecystectomy**.

Gallstones + Asymptomatic → Reassure.

CBD stones ± Asymptomatic → Laparoscopic Cholecystectomy.

So, in both conditions, first-line investigation → Ultrasound.

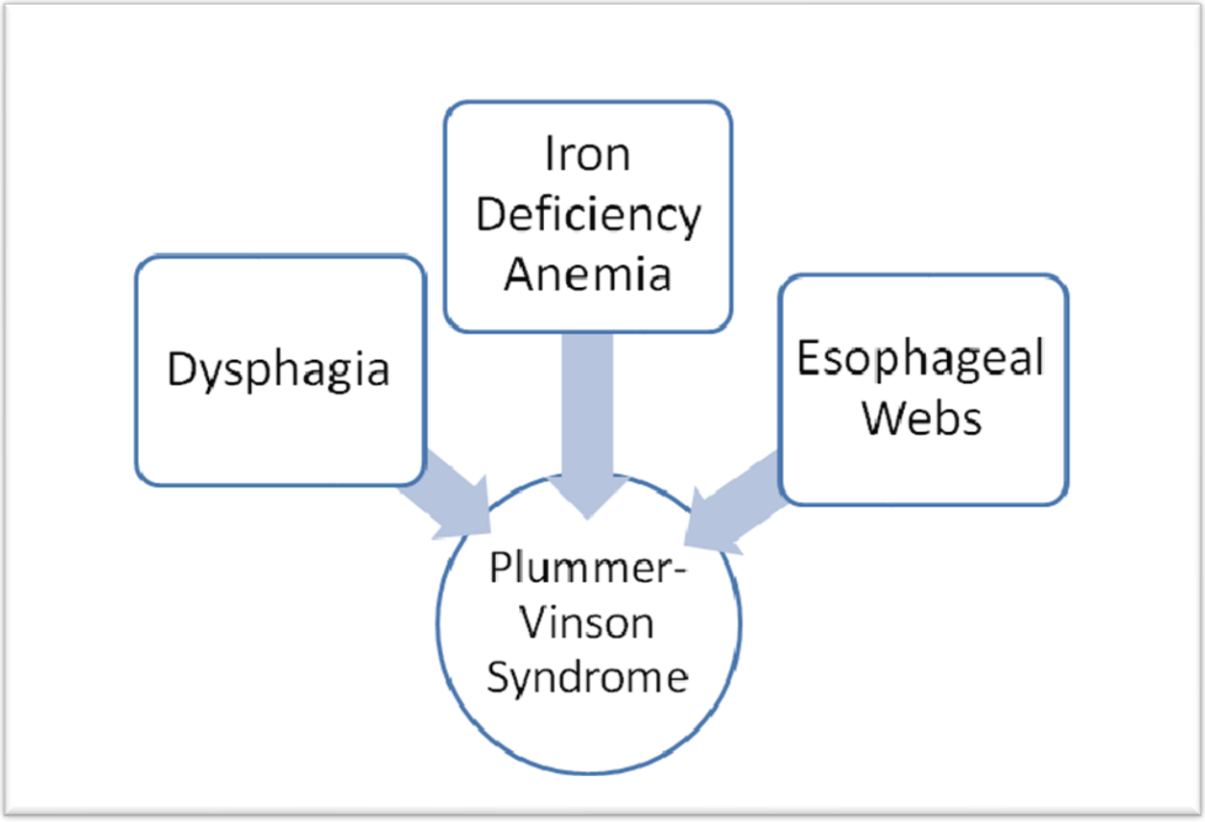


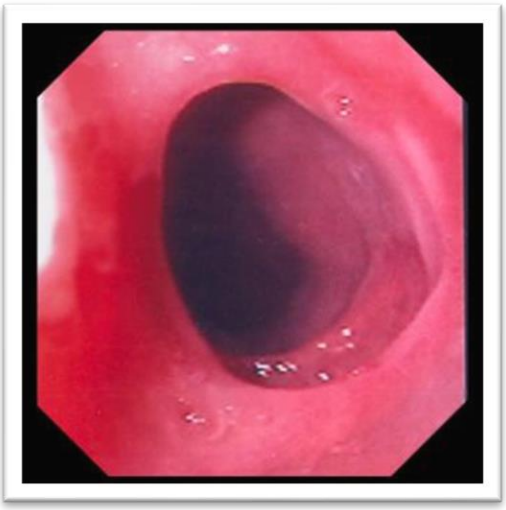
The Biliary Tree

Key 15

Plummer Vinson Syndrome

- **Post-cricoid Oesophageal Webs → *Painless, intermittent Dysphagia.***
- **Iron Deficiency Anemia → *Pallor, Fatigue, Glossitis, Angular stomatitis, Koilonychia.***
- **℞ → Iron Supplements + Webs Dilatation**



	 **Oesophageal Web** **In Plummer Vinson Syndrome**
Key 16	• 2 important medications to remember: Alendronate "**Bisphosphonate** used in osteoporosis" and **NSAIDs**. Both can worsen Oesophagitis and Gastro-Oesophageal Reflux Disease "GERD" Leading to → Scars of the oesophagus → (**Benign Oesophageal stricture**). This condition is characterised by **PERSISTENT Dysphagia** to both solids and fluids **WITHOUT** "Regurgitation". • Slowly **progressive Dysphagia** in a young adult. No Weight loss. Hb is normal. Hx of taking **H2-blockers** (e.g. Ranitidine) for **retrosternal discomfort** (**GERD**) for long period → think of a benign stricture (e.g. **peptic stricture**). • (**Barret's Oesophagus**) is similar to Benign Oesophageal Stricture. However, the dysphagia is **OCCASIONAL**, not persistent.

	• "**Regurgitation**" is an alarming word for (**Achalasia**: *Bird Beak Appearance*) and (**Pharyngeal Pouch**). √ However, the latter "**Pharyngeal Pouch**" has other specific features such as **Halitosis** "*bad breath smell*", ***Stale food regurgitation***, gurgling sound when swallowing fluids, a **sensation of a lump in the throat**, neck bulge. √ In **achalasia**, besides regurgitations, the patient may have frequent chest infections due to regurgitations "aspiration pneumonia" ± the dysphagia may be progressive "worsening with time". **Example Scenario** **An elderly patient presents with Hx of Persistent Dysphagia to both solids and liquids. No Weight Loss. No Regurgitation. He takes Alendronate for his Osteoporosis. What is the Dx?** → **Benign Oesophageal Stricture** (PERSISTENT dysphagia, Alendronate, No regurgitation)
Key 17	**Scenario:**

	23 Y/O Female. Lethargy, Weigh Loss, Abdominal Discomfort, Bloody Diarrhea > 8 times a day. Endoscopy → **Deep Ulcers** and **Skip Lesions** → **Crohn's Disease** Although bloody diarrhea is more common with UC than CD, the endoscopic findings of Skip Lesions and Deep "transmural" ulcer are pathognomonic for CD.
Key 18	**Acute Pancreatitis** • Hx of: **Gallstones**, **Alcoholism**, **Trauma**, **ERCP**. • **Upper abdominal pain** that radiates to the **back** and relieves by **sitting** or **leaning forwards**. • + **Nausea**, **Vomiting** • **± Tenderness, Tachycardia, Shock, Periumbilical bruising (Cullen's sign), Jaundice.** ◙ **Investigation** →

√ **Initial** → Serum **Lipase** and **Amylase: (Lipase is more specific and sensitive)**

↑ serum Lipase more than 3 times the upper normal limit.

√ **To confirm** → **CT with contrast** of the Pancreas.

◙ Management

◙ initially, the BEST step is Supportive:

IV fluid "Fluid resuscitation"

+ **Analgesics**

+ **Nutritional support**

◙ Then →

IV antibiotics (e.g. IV imipenem) in moderate to severe cases

◙ Surgical debridement is done only if there is evidence of necrosis. It uses minimally invasive procedures such as (transgastric endoscopy and video-assisted translumbar retroperitoneal necrosectomy).

Acute Cholangitis "Ascending Cholangitis"

- *Charcot's Triad* (**FRJ**)

  Fever,

	Right upper quadrant pain, **Jaundice**. ± **HL** (HypOtension and Leucocytosis). • ***Investigations*** → Ultrasound and Blood cultures. • ***Management:*** √ Fluid resuscitation. √ Broad-spectrum intravenous antibiotics. √ Correct any coagulopathy. √ Early ERCP.
Key 19	**Notes on Organisms Causing Diarrhea** • Traveller's diarrhea that is usually of a **short period** and **self-limiting** (especially Hx of a travel to **Africa**) → **E. coli.**

- Hx of travel to **Europe**, **WATERY** (Non-bloody) diarrhea, Weight Loss (If chronic Giardiasis), abdominal pain and bloating (Symptoms for **> 10 Days**) → **Giardia**.

- Hx of travel → **Prodrome**: **HIGH Fever (40 C)**, **Headache**, **Myalgia** → Followed by **BLOODY Diarrhea** → **Campylobacter jejuni**.

- ***BLOODY DIARRHEA Organisms (CSS) →***

  ***C**ampylobacter*, ***S**higella*, ***S**almonella*.

Other Notes:

The most common:

- Bloody diarrhea → **Campylobacter jejuni** → Followed by **Shigella**.
- Traveller's diarrhea → **E. coli**.
- Diarrhea in Paediatrics → **Viral** (**Rota Virus**).
- Diarrhea + Weakness + Areflexia → **Guillain-Barre Syndrome**.

	○ Diarrhea + Renal Impairment + Hemolysis → **Hemolytic Uremic Syndrome (HUS)**. ○ Diarrhea followed by RUQ Pain → **Amoeba**. ○ Watery Diarrhea after camping or long travel in Europe → **Giardia**. ○ Diarrhea after long-term antibiotics → **Clostridium Difficile** (**Pseudomembranous colitis**) Rx → **Oral Vancomycin "1st line"**. or **Oral Metronidazole "2nd line".** ○ Diarrhea after eating Eggs or Chicken → **Salmonella**. ○ Diarrhea just hours after a meal → **Staph**. **Toxin**. ○ Diarrhea in a bedridden-patient (eg, handicapped) with stony hard stools → **Fecal impaction**.
Key 20	**Scenario:** A Mass has been removed from the **Cecum** **(RIGHT Side)** The histopathology report → **Transmural** infiltration with lymphocytes and **granulomas** without necrosis. The Diagnosis is → **Crohn's Disease**. **Notes on Crohn's Disease:** • CD usually starts at the **ileocecal junction** (Right iliac fossa).

	• Histology of CD → ↑ **Goblet** Cells, **Granuloma**, **Transmural**. • Endoscopy of CD → **Skip lesions**, **Cobblestone** appearance, **Deep Ulcers** (Transmural) • Small bowel enema → **Rose thorn ulcer**, **Kantor's string sign**, **Fistulae**.
Key 21	**Scenario:** 55 Y/O woman. Severe abdominal pain radiates to the back for 1 day. + Nausea and Vomiting and Tachycardia and looks in shock. No fever or Diarrhea. Hx of **gallstones**. **The most likely Dx →** **Acute Pancreatitis**. **An investigation to confirm the Dx →** **Serum Lipase** (X 3 folds) **Notes:** o Serum lipase in more sensitive and more specific than serum amylase in acute pancreatitis. o The most common causes of Acute pancreatitis in the UK → **Gallstones** and **Alcohol.**

	○ Other Causes → **ERCP**, **Trauma**, **↑ Triglycerides**.
Key 22	**Young** age, **Diarrhea**, sometimes **bloody**, Chronic **Abdominal pain**, **Tenesmus** → Think of **Inflammatory Bowel Disease** **(UC/CD)**. ○ **Why not Diverticulosis?** → Diverticulosis is usually **Asymptomatic** unless complicated. ○ **Why not Irritable Bowel Syndrome?** → IBS does not have **Bloody** diarrhea.
Key 23	**Chest pain, *Difficulty in swallowing both liquids and solids*, Recurrent chest infections** → **Achalasia** ○ Dysphagia to both liquids and solids. ○ Recurrent chest infection → mainly due to Regurgitation (seen in Achalasia) → Aspiration pneumonia.

Key 24	In patients with **Oesophageal cancer with liver metastasis** → No surgery. End-stage oesophageal cancer is inoperable. **So, how to relieve the symptom of Severe Dysphagia?** → **Stenting "endoluminal stenting"** Insert a stent into the oesophagus, it will expand and open the obstruction (Fast and Effective).
Key 25	**Anemia a few years after gastrectomy, MCV is 110, Loss of proprioception and vibration sense. There are also fatigue, exertional dyspnea and palpitations. What is the Diagnosis?** → **Vitamin B12 deficiency**. • **Gastrectomy** → Loss of the intrinsic factors in the stomach → impaired Vitamin B12 (Cobalamin) absorption. • After gastrectomy, there are **Cobalamin stores** that may take up to **1-2 years** to deplete. After that, Vit B12 deficiency develops. • In Vit B12 deficiency: High MCV (Macrocytic Anemia), Hypersegmented neutrophils, Peripheral paraesthesia, Loss of position, vibration and proprioception sense.

	• Fatigue, Exertional Dyspnea, Palpitations → **Anemia symptoms**. • Rx → Intramuscular Hydroxocobalamin.
Key 26	***Chronic diarrhea***, ***Weight loss***, ***Perianal fistulas***. → Think of **Crohn's Disease**. • **Weight Loss** is more common in CD than in UC. • **Diarrhea without blood** is more common in CD than in UC. • **Fistulas** are more common with CD not UC. "Remember, in CD, there are deep ulcers -Transmural- ".
Key 27	• Left Supraclavicular Mass → Virchow's Node → Indicative of **Gastric Carcinoma** (Anorexia, Dyspepsia, Weight Loss, Old age). This sign is called → **Troisier's sign**. • Right Supraclavicular Mass → **Oesophageal cancer**, **Lung cancer**, **Hodgkin's Lymphoma**.

	• **Pancoast Tumour** → A tumour of the **Apex of the Lung** (at the top end of either the left or the right lung). It typically spreads to the nearby tissues such as the ***Ribs*** and the ***Vertebrae***. Most Pancoast tumours are Non-small cell lung cancer.
Key 28	**PriMary Biliary Cirrhosis** • **3 Ms:** o **Anti-Mitochondrial Antibodies.** o **Middle aged-Female.** o **IgM** • **Others:** o **Pruritus** = Skin Excoriations. o **↑ Alkaline Phosphatase**. o **Jaundice**. o Common association → **Sjogren's Syndrome**. **Primary Sclerosing Cholangitis** o Diagnosed by → **ERCP.** o Common association → **IBD (**particularly **Ulcerative Colitis). Imp √** o The others are more or less similar: **Pruritus, Jaundice, ↑ ALP.**

	◙ **The treatment in both conditions →** Ursodeoxycholic acid, cholestyramine. --- **Important Differentials:** ◙◙ Middle-aged Female + Abnormal LFTs + 2ry Amenorrhea + Presence of autoimmune disease (e.g. hypothyroidism) → **Autoimmune Hepatitis**. ***(ALP is normal or mildly ↑).*** ◙◙ Hx of heavy alcohol consumption + Signs of liver disease/ cirrhosis: Ascites, Hematemesis, Jaundice, Hepatomegaly, Spider naevi. + **↑ AST:ALT ratio** (e.g. AST:150, ALT: 70). + **(GGT) is also increased** → **Alcoholic Liver Disease**.
Key 29	**Case Scenario:** An elderly with multiple liver metastasis 2ry to colon cancer. Drowsy, cachexic.

Abdominal pain, Jaundice.

Ascites and LL Oedema.

Low urine out-put (Oliguria).

High ALP, High Bilirubin

Low Albumin, Mildly Low Sodium (128)

Normal Urea, Creatinine, Potassium and Calcium.

What is the most appropriate treatment?

→ **Albumin Infusion**

Albumin infusion → **↑ Oncotic (Colloid Osmotic) pressure** → Shift of the fluids from extravascular to intravascular compartments → Alleviation of Ascites and Oedema + Good Perfusion to the Kidneys and thus restoration of normal urine-output.

- N.B. Haloperidol can be used in small doses as an anti-emetic.
- N.B. Haloperidol can cause Hyponatremia.

A differential:

In ascites secondary to Liver cirrhosis

	◙ **Give** → **Spironolactone**. √ imp. ◙ **Giving** → **Albumin infusion** is also beneficial. √ imp.
Key 30	**Celiac Disease** o Autoimmune, Malabsorption disease, results due to sensitivity to the **Gluten** (which is a protein). o Eating gluten diet (e.g. **Rye**, **Wheat**, **Barley**) → **Villous atrophy** of the GIT → Malabsorption → Iron deficiency Anemia, Folic Acid and Vit. B12 Deficiency, malabsorption of fat. o **Manifestations:** • Chronic or Intermittent **Diarrhea**. • **Steatorrhea** (fatty stools due to malabsorption of fat). • **Stinking,** bad-smell **stools** • **Abdominal discomfort, Bloating, Nausea and Vomiting.** • **Wight Loss.**

- **Iron deficiency anemia** (the most common), followed by **Folate deficiency** then **Vit B12 deficiency**.
- Manifestations of anemia e.g. **Fatigue**.

- **Complications**: ***Osteoporosis*** / ***T-cell lymphoma*** (rare).
- **Association not to be forgotten → Dermatitis Herpetiformis.**

- **Diagnosis:**

- Positive **TTG** and **IgA**. (First Line)

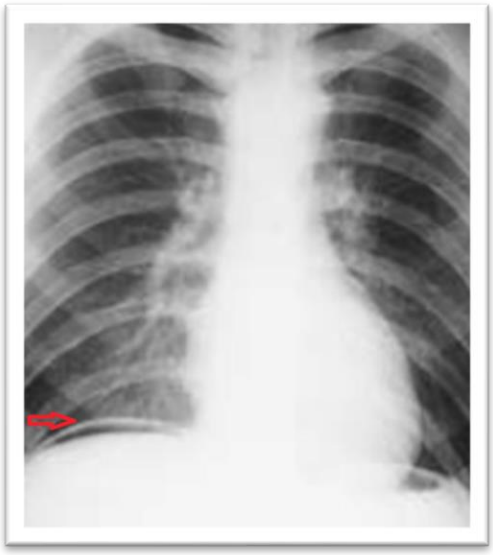
  (TTG= **Tissue TransGlutaminase Antibodies**)
- Positive Endomysial Antibodies.

If TTG is positive, we need to confirm the diagnosis of Celiac disease by a Biopsy →

- **Jejunal or Duodenal Biopsy:**
- **Villous Atrophy.**
- **Crypt hyperplasia**.
- ↑ inter-epithelial **lymphocytes**.

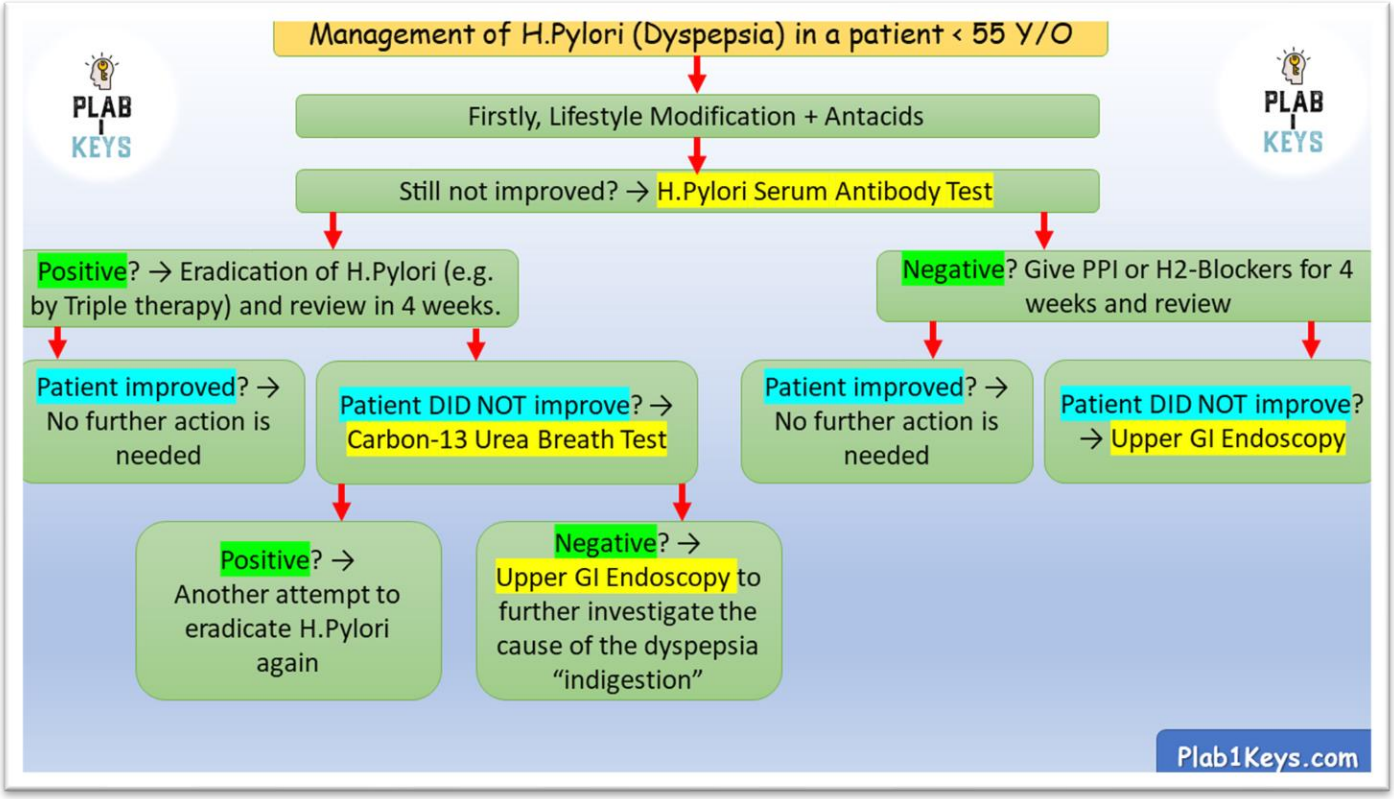
	Important: for the biopsy to be accurate, the patient should re-introduce the gluten in his diet for 6 weeks before the biopsy. o **Treatment** → **Gluten-free diet**. **Example scenario:** 33 Y/O male, Non-smoker. Recurrent and Chronic Diarrhea for 6 Months. His clothing appears to be ill-fitting (indicative of weight loss). Hb=11 MCV= 105 (high) o The most likely Diagnosis → **Celiac Disease**. o Endoscopy + Duodenal Biopsy will show → **Villous Atrophy**.
Key 31	**Scenario on Celiac Disease** 50 Y/O male. A Known case of Celiac disease since childhood. Recently developed diarrhea and weight loss.

	Think of a complication of celiac → **intestinal lymphoma** (T-cell lymphoma is a rare complication of the celiac disease). ***Remember the following conditions that may develop on top of celiac disease:*** 1) Iron deficiency Anemia. (The commonest Anemia) 2) Folic Acid Deficiency. (The 2nd Commonest Anemia) 3) Vitamin B12 Deficiency. (The 3rd Commonest Anemia). 4) Osteoporosis. 5) T-Cell Lymphoma (Intestinal Lymphoma). (Rare) 6) Dermatitis Herpetiformis (Skin Rash). 7) DM Type 1.
Key 32	**Treatment of Acute Cholecystitis.** o In a **stable** patient → **Laparoscopic Cholecystectomy**. o In **a non-stable** patient (e.g. high temperature, severe tenderness, low blood pressure due to *Gallbladder perforation*) → **Emergency Laparotomy**. o Incidental finding of **Gallstones** in an **Asymptomatic** patient

	→ Reassurance. o Incidental finding of **CBD stones** in an **Asymptomatic** patient →ERCP or Laparoscopic cholecystectomy. "something has to be done"
Key 33	**A Study Scenario** √ **Sudden** onset of **Severe Abdominal Pain** √ Develops to **severe generalised abdominal pain and tenderness** √ **ill-looking patient** (*lies motionless*, *diaphoretic*, with shallow rapid breathing) √ Abdominal tenderness and guarding √ **Erect "47pright" X-ray** shows → **Free air under diaphragm**. "however, the diagnosis is not excluded if no air under diaphragm is seen"  • **Diagnosis** → **Perforated Peptic Ulcer**. • **Treatment** → ▫ ***Nil by Mouth (NBM): give IV fluids, IV antiemetics (e.g. metoclopramide 10 mg), IV analgesics, IV antibiotics*** ▫ Refer for **Urgent Surgery** to correct the perforation.

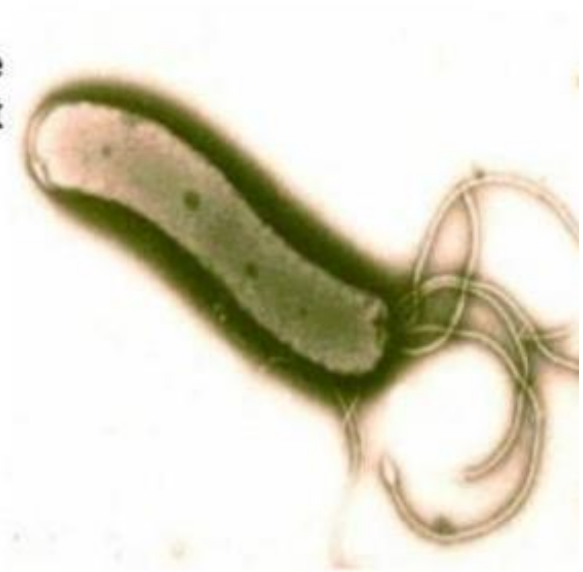
Key 34	**H. Pylori Management (chronic dyspepsia, indigestion)** • Helicobacter pylori is a Gram-negative bacterium associated with a variety of gastrointestinal problems, principally peptic ulcer disease. **In addition to the following diagram, remember these notes:** • **Triple therapy** for eradication of Helicobacter Pylori → **Proton pump inhibitors** (e.g. omeprazole, esomeprazole) + **Amoxicillin** + **Clarithromycin** (7-14 days). • **Example of Triple Therapy: (for 7 to 14 days)** o **Esomeprazole (Proton Pump Inhibitor)**: 20 mg BID o **Amoxicillin**: 1-gram BID o **Clarithromycin**: 500 mg BID • **PPI** should be stopped **14** days before testing for H. Pylori. • **Antibiotics** should be stopped **28** days before testing for H. Pylori.

- After a full course of H. Pylori treatment, we cannot re-test the eradication of H. Pylori using serological test. This is because the antibodies stay for long time after a successful eradication**.**



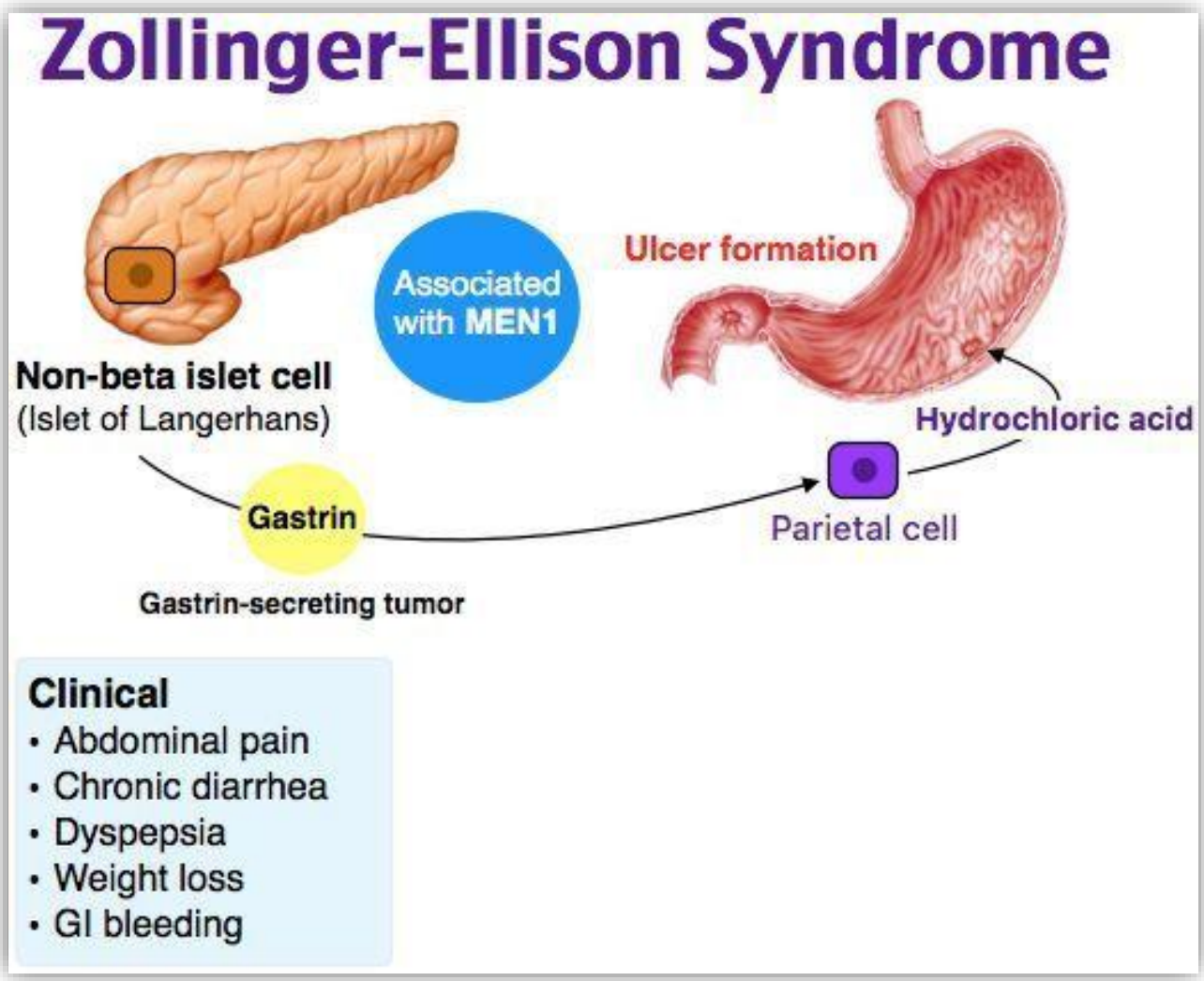
- The most important note to remember is that in a treated patient of H. Pylori with persistence of the symptoms → we do **C-13 Urea breath Test**

  (Not C-14 Nor Stool antigen test).
- Also, the most appropriate test to ensure successful eradication of H.Pylori is → **Carbon 13 Urea breath Test**
- What if C 13 test is not available? → stool antigen test.

	• Manifestations of H. Pylori are overlapping, not specific: **Symptoms of H.pylori infection** • Abdominal pain with burning or gnawing sensation. • Pain is often made worse with empty stomach; night time pain is common. • Poor appetite. • Weight loss. • Heart burn. • Indigestion (dyspepsia) • Belching. • Nausea. • Vomiting. • Blood in stool.  **NOTE**: √ ≥ 55 YO (+) Dyspepsia (+) Weight loss → "urgent" upper GI endoscopy to exclude oesophagel/ gastric cancer.
Key 35	• Ulcers at **various** and unusual **sites** that are **resistant** to treatment → Think of **Zollinger-Ellison Syndrome**.

Zollinger Ellison Syndrome

- Characterised by the presence of tumours (**Gastrinomas**) in the **duodenum** or in the **pancreas**.
- Gastrinoma releases excessive amounts of **Gastrin** which stimulates the parietal cells of the stomach to release more and more hydrochloric acid → **multiple ulcers**
- The ulcers are often **resistant to treatment** and present on **unusual sites** (eg, oesophagus, distal duodenum, jejunum).
- They tend to recur after adequate treatment or surgery.
- Due to the excessive hydrochloric acid released by the stomach, there might be associated **Watery or Fatty Diarrhea**.
- **Diagnosis** → **Fasting Gastrin Level** (OR) **Secretin Stimulation Test**.



Example scenario

A patient has recently received a full course for H. Pylori eradication. He now attends with burning retrosternal pain for a few days. Endoscopy is done and it shows multiple ulcers in the lower oesophagus, stomach and duodenum. What should be done next regarding the investigation?

	→ either **Fasting Gastrin Level** "best" (OR) **Secretin Stimulation Test** √ Do not get tricked and pick C-13 Urease test! √ Note that an endoscopy is already done and shows that "Multiple" Ulcers are seen although he had previously been treated. (***Refractory or Resistant Multiple Ulcers on Unusual sites***) → Think of **ZES** and order Fasting **Gastrin** Level.
Key 36	**Treatment of Ulcerative Colitis** **To Induce Remission:** • **Mild to Moderate UC** → Rectal **Mesalazine** → (If not responding then Oral Mesalazine). • **Severe UC** → Admission and **IV hydrocortisone**. **To maintain Remission:** • Oral Mesalazine (If does not work → Oral Azathioprine or Oral Mercaptopurine) ***Note: Mesalazine is 5-ASA (Aminosalicylic Acid)***

Treatment of Crohn's Disease

- **To Induce Remission** → **Oral Prednisolone** (First line). Second is budesonide. If both are not in the option, pick Mesalazine (2nd line).
- **To Maintain Remission** → Oral Azathioprine or Mercaptopurine.

When can we decide that it is SEVERE Ulcerative Colitis?

The rule of **6**, **30**, **90**

- > **6** bowel movements a day + Visible blood in large amounts.
- ESR > **30** (also WBC may be elevated).
- HR > **90** "mildly tachycardic"
- Temperature > 37.8 "mild feverish"
- Anemia (low Hb): (Pallor, Fatigue …etc).

→ Admit and give **IV Hydrocortisone**

Key 37

Scenario:

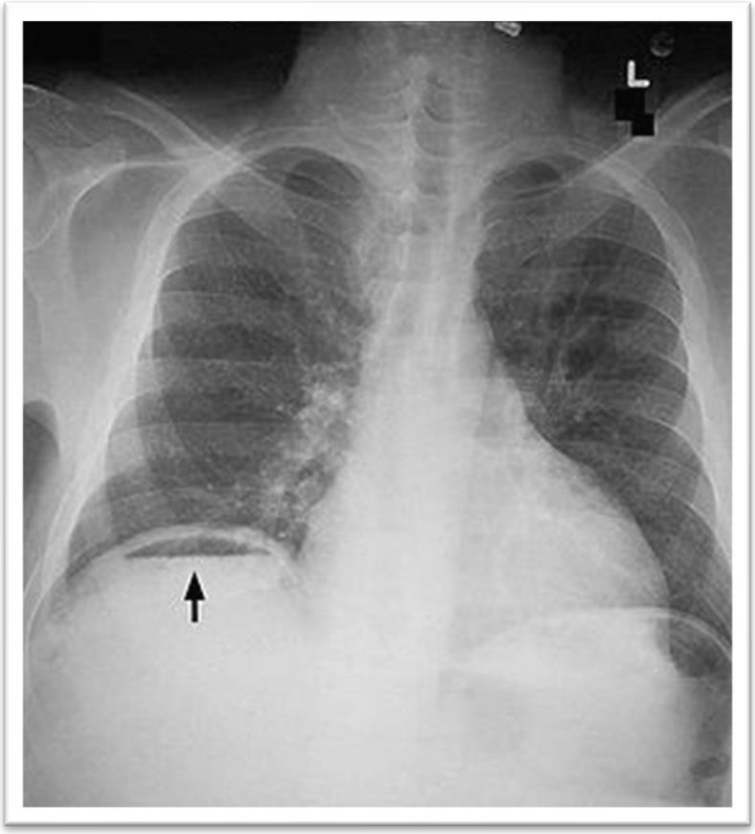
Hx of indigestion. Long Hx of Ibuprofen (NSAIDs) intake. Sudden onset severe Abdominal pain. Abdominal Rigidity, Absent Bowel Sounds, Lying motionless.

Dx? → **Perforated peptic ulcer**.

Investigations:

- Useful → **Erect Chest X-ray** → **as it would show air under Diaphragm**.
- The **primary** (**Diagnostic**) Investigation → **Upper GI Endoscopy**. √

(It can diagnose and look for the source of bleeding and stop it)!



	Treatment? → IV fluids, Analgesics, Antiemetics, Antibiotics (**All IV**). → Refer for **Urgent Surgery**.
Key 38	The treatment for celiac disease is → **Gluten-Free Diet**. **Remember!** ***Before performing investigations for Celiac disease*** ***(e.g. Tissue Transglutaminase antibodies / Jejunal or Duodenal Biopsy*** → **Restart Gluten intake for 6 weeks before testing**.
Key 39	**Hereditary Haemochromatosis** o Autosomal Recessive. o ↑ Intestinal Absorption of Iron → **Iron Accumulation "Deposition" in Tissues**, such as: • **Liver** "The main organ of iron deposition" → **Hepatomegaly**, **Cirrhosis** → **Hepatocellular carcinoma = Hepatoma** "Hepatic Cancer". • **Pancreas** → **Diabetes Mellitus**. • **Skin** → **Bronze Skin (Hyperpigmentation)**.

	• **Joints** → **Arthropathy**. • **Heart** → **Arrhythmia**, **Cardiomyopathy**, **murmur**, **SOB**. **Remember the triad**: **Hepatomegaly** + **DM** + **Bronze Skin** **Remember** that the "**Liver**" is the most likely organ to develop cancer in Haemochromatosis (Due to Cirrhosis and iron deposition). **Scenario:** 40 Y/O Male. Fatigue and SOB. DM and Hepatomegaly. High Ferritin level. • The most likely Diagnosis → ***Haemochromatosis***. • The most likely organ to get cancer → ***Liver***
Key 40	**Scenario:** 55 Y/O Male. Skin Hyperpigmentation. Spider Angioma. Hepatomegaly. Heart murmur. Mild joint pain. Diagnosed as Restrictive Cardiomyopathy. • The most likely Diagnosis → **Haemochromatosis**. • He is at an Increased Risk of → **Hepatoma (HCC)**.

	○ Deposition of the iron in the **liver** → Hepatomegaly → later on Cirrhosis → Risk of HCC (Hepatoma). ○ Deposition of the iron in the **skin** → skin Hyperpigmentation. (bronze skin). ○ Deposition of iron in the **heart** → Cardiomyopathy. ○ Deposition of iron in the **joints** → Arthropathy.
Key 41	**Severe epigastric pain that radiates to the back and relieved when sitting forwards** **+** **Vomiting** **+** **Hx of Gallstones (OR) Alcohol intake (OR) ERCP (OR) Trauma.** → Think of **Acute Pancreatitis**. → Order: **Serum Lipase**.
Key 42	• **Persistent dyspepsia** (Severe Recurrent Epigastric Pain especially at night) in a patient who **has never had any investigations** or treatment for it.

	○ **The best "Next" "Initial" Step** → **H. Pylori Serology Test**. ○ **The best investigation that would lead to the diagnosis** → **Endoscopy**.
Key 43	• In addition to the **common features of Vit B12 Deficiency** (**Peripheral Paraesthesia, Impaired proprioception, position and vibration sense**), there are other **uncommon features** such as (lemon-yellow tinge of the skin and glossitis). • N.B. even **partial** not total **gastrectomy** can lead to Vit B12 deficiency in the long-run.
Key 44	**Chronic diarrhea, recently becomes bloody, + aphthous ulcer + Left Lower Quadrant Tenderness + No weight loss.** **The likely Dx** → **Ulcerative Colitis.** ○ **Bloody diarrhea** → in favour of UC. ○ **Aphthous ulcers** → In both UC and CD (more in CD). ○ **Left Lower Quadrant** → in favour of UC. (in CD→ Right iliac fossa).

	○ **Weight loss** is more prominent with CD. Here, there is no weight loss; therefore, UC is more likely the diagnosis.
Key 45	**Chronic diarrhea + Abdominal discomfort + Weight Loss + Endoscopy shows cobblestone mucosa.** **The likely Dx** → **Crohn's Disease**.
Key 46	**Scenario** 42 Y/O Male. Dysphagia to both solids and liquids. Occasionally, there is severe retrosternal pain. Barium Swallow → Dilated Oesophagus that tapers toward the distal end. ○ **Diagnosis** → **Achalasia**. ○ **Treatment** → **Dilatation of the lower Oesophageal Sphincter**. **Achalasia** • **Inability to relax the lower oesophageal sphincter (LOS)** due to an idiopathic loss of the normal neural structure.

	i.e. (↑ Lower Oesophageal ***Resting*** Pressure). • **Investigations:** o **X-ray** → **Megaoesophagus** = Large or dilated oesophagus. o **Barium meal** → **Bird's Beak** Appearance of the distal end of the oesophagus. (**Dilated Oesophagus that tapers -Narrows- Towards the distal end of the oesophagus**) o **The most accurate** → **Oesophageal Manometry** → **Increased lower oesophageal *resting pressure***. o **Rx** → **Dilatation of the lower oesophageal sphincter**.
Key 47	**Scenario** 40 Y/O Woman. Pruritus, Jaundice, ↑ ALP, a Known case of Sjogren Syndrome. **Investigation → Anti-Mitochondrial Antibodies (AMA)** **Diagnosis → Primary Biliary Cirrhosis**

PriMary Biliary Cirrhosis

3 Ms

- **Anti-Mitochondrial Antibodies.**
- **Middle aged-Female.**
- **IgM**

Others

- **Pruritus** = Skin Excoriations.
- **↑ Alkaline Phosphatase**.
- **Jaundice**.
- Common association → **Sjogren's Syndrome**.

Primary Sclerosing Cholangitis

- Diagnosed by → **ERCP.**
- Common association → **IBD (**particularly **Ulcerative Colitis).**
- The others are more or less similar: **Pruritus, Jaundice, ↑ ALP.**

	The treatment in both conditions → Ursodeoxycholic acid, cholestyramine. **Remember that:** Middle-aged Female + Abnormal LFTs ± 2ry Amenorrhea ± Autoimmune disease (e.g. thyroid disease, vitiligo, DM 1). → **Autoimmune Hepatitis**. **(ALP is normal or mildly ↑).**
Key 48	• **A patient with a Hx of peptic ulcer is prone to get perforated peptic ulcer after surgery. Why?** o This is because the **stress** caused by the operation leads to a **high production of Gastric Acid** → Perforated peptic ulcer. **Scenario:** A patient with Hx of peptic ulcer underwent surgery to remove the head of the pancreas due to cancer. A few days later, he develops abdominal pain with rigidity, tenderness and guarding. The vitals are as follows: mild fever, hypotension, tachycardia. o **The likely diagnosis →** **Perforated peptic ulcer** (Acute Abdomen, Shock)

	○ **The next step →** **Erect chest and abdomen X-ray** (air under diaphragm). ***N.B., We perform Erect chest and abdominal x-ray to look for air under diaphragm seen in perforated peptic ulcer. Not U/S nor CT scan!***
Key 49	In a patient with **Positive IgA** and **Tissue Transglutaminase** (suggestive of **Celiac Disease**), we still need to confirm the diagnosis by (**Jejunal or Duodenal Biopsy**) before advising the patient to eliminate the gluten from their diet. **Scenario** A DM type 1 patient with intermittent diarrhea, Abdominal bloating, tiredness over the last few months was found to have Positive Tissue Transglutaminase Antibodies. What is the next step? → Confirm the diagnosis of celiac disease by **Jejunal/Duodenal biopsy** before advising the patient to go on a gluten-free diet. **Remember that, Celiac disease can be associated with:** 1) Iron deficiency Anemia. (The commonest Anemia)

	2) Folic Acid Deficiency. (The 2nd Commonest Anemia) 3) Vitamin B12 Deficiency. (The 3rd Commonest Anemia). 4) Osteoporosis. 5) T-Cell Lymphoma (Intestinal Lymphoma). (Rare) 6) Dermatitis Herpetiformis (Skin Rash). 7) DM Type 1.
Key 50	***A patient came from Kenya (a country in Africa) develops watery diarrhea with abdominal cramping.*** **The most likely organism → E. Coli** • Traveller's diarrhea that is usually of a **short period** and **self-limiting in 72 hours** (especially Hx of a travel to **Africa**) → **E. coli** • Hx of travel to **Europe**, **WATERY** (Non-bloody) diarrhea, **Weight Loss** (If chronic), abdominal pain and bloating (Symptoms for **> 10 Days**) → **Giardia**

	• Hx of travel → **Prodrome "initially"**: **HIGH Fever (40 degrees)**, **Headache**, **Myalgia** → Followed by **BLOODY Diarrhea** → **Campylobacter jejuni**.
Key 51	**Scenario**: 27 Y/O Female with a 1-week Hx of Jaundice, fever, malaise. She has recently been diagnosed with hypothyroidism and takes levothyroxine. LFTs are abnormal ALP is 210 (Normal: 30-150). Diagnosis → **Autoimmune hepatitis**. Middle-aged Female + Abnormal LFTs ± 2ry Amenorrhea ± Autoimmune disease (e.g. thyroid disease, vitiligo, DM 1) → **Autoimmune Hepatitis** ***(ALP is normal or mildly ↑).*** • **Normal Alkaline Phosphatase is 30-150. If there is increase > 300, think of another diagnosis than autoimmune hepatitis.** **Important Differentials of Liver Disease** o **1ry Biliary Cirrhosis → Middle-aged female, Pruritus, Jaundice,**

↑ ALP, associated with Sjogren's Syndrome.

Investigation: Anti-Mitochondrial Antibodies.

- 1ry Sclerosing Cholangitis → the same but the association is usually IBD (mainly Ulcerative colitis).

  Investigation: ERCP

- Autoimmune hepatitis → Early-middle aged female, abnormal ALT and AST, Normal or mildly elevated ALP ± 2ry Amenorrhea ± another autoimmune disease (e.g. hypothyroidism, vitiligo, rheumatoid arthritis, celiac, pernicious anemia)

- Alcoholic Liver Disease → Hx of heavy alcohol consumption. Signs of liver disease/ cirrhosis: Ascites, Hematemesis, Jaundice, Hepatomegaly, Spider naevi. Both AST and ALT are elevated; however, AST is more elevated than ALT: ↑ AST:ALT ratio (e.g. AST:150, ALT: 70). Gamma Glutamyl Transferase (GGT) is also increased.

- Cholestatic hepatitis → Old age, Hx of a recent use of co-amoxiclav, amoxicillin, flucloxacillin "drug-induced hepatotoxicity".

  Features: dark urine, itchiness "pruritis", fatigue, nausea, vomiting, abdominal pain.

  Labs: ↑ ALT, ↑ ALP "alkaline phosphatase", ↑ GGT, ↑ bilirubin.

Key 52	**Scenario:** 22 Y/O Female 4 months of intermittent diarrhea and abdominal bloating. Blistering rash on her elbow. Low Hb. Duodenal biopsy shows shortening of the villi + Lymphocytosis. Diagnosis → **Celiac disease** • Blistering rash on the elbow → **Dermatitis Herpetiformis** (a skin lesion commonly linked to Celiac disease) • Shortening of the villi = **Villous atrophy** • **Remember that in Celiac disease:** o Positive TTG (**Tissue Transglutaminase**) "First Line". o Duodenal/Jejunal biopsy → **Villous atrophy**, **Crypt hyperplasia**, **lymphocytosis**.

Key 53	**Clostridium difficile [Pseudomembranous Colitis]** • **Receiving certain Antibiotics can suppress the normal flora that inhabits the GIT. Therefore, C. Difficile becomes free to infect the GIT causing "Pseudomembranous Colitis".** • **<u>Examples of the antibiotics that can cause C. Difficile:</u>** Clindamycin, Amoxicillin, Ampicillin, Co-Amoxiclav, Broad spectrum cephalosporin, Quinolones (e.g. Ciprofloxacin). • **<u>Manifestations:</u>** o Hx of recent treatment with antibiotics (e.g. Amoxicillin, clindamycin) o Diarrhea (might be bloody). o Abdominal pain (might be very severe). o Fever. o High WBCs and CRP.

	• **Investigation** → Clostridium Difficile Toxin (CDT) in the stools. • **Treatment**: o 1st Line → **Oral Vancomycin**. o 2nd line → **Oral Metronidazole**. (Recently, **vancomycin** has become the first line. However, if it is not in the options, pick the second line which is metronidazole). **Scenario** A patient with Cellulitis admitted for 3 days and treated with clindamycin. Soon after, he develops bloody diarrhea, abdominal pain and high fever. WBCs and CRP are high. • The diagnosis? → **C. Difficile (Pseudomembranous Colitis)**. • The treatment → **Oral Vancomycin**. • If not on the options? →pick **metronidazole**.
Key 54	**Scenario** 40 Y/O male with type 2 DM. Complains of pain in his joints, increase in breast size, fatigue.

	O/E: deep tan coloured skin, enlarged liver. Diagnosis? → **Haemochromatosis** ***Autosomal recessive, accumulation of iron in the tissues*** **Liver** → **hepatomegaly**, **cirrhosis** → risk for **cancer (HCC = Hepatoma).** **Heart** → **arrhythmia**, **cardiomyopathy**. **Skin** → **bronze skin** / **deep tan skin** / **skin hyperpigmentation**. **Joints** → **arthropathy** **Pancreas** → **DM**.
Key 55	***40*** Y/O ***Obese female*** + ***Severe upper abdominal pain*** + ***Vomiting*** + ***Fever*** and ↑ ***WBCs*** (inflammatory element) ◙ **The likely Dx** → **Acute cholecystitis**. √ Remember: 5-F Syndrome—**fair**, fat, **female**, fertile and over forty. √ It may present with either upper abdominal or right hypochondrial pain.

	◙ **Investigation** √ **Ultrasound**: first-line investigation of choice: Thick walled and Shrunken Gallbladder √ If the diagnosis remains unclear then cholescintigraphy (HIDA scan) may be used. ◙ **Treatment** √ Intravenous antibiotics √ NICE now recommend **early laparoscopic cholecystectomy, within 1 week of diagnosis**. Previously, surgery was delayed for several weeks until the inflammation has subsided.
Key 56	**Scenario** 24 hours after ERCP (Endoscopic Retrograde Cholangiopancreatography) for gallstones, a 40-year-old female returns to the A&E complaining of severe right upper quadrant pain and tenderness, high fever (38.9 degrees) and she looks yellow. Bilirubin WBCs and CRP are elevated. ALT and AST are normal. Amylase is 300 U/L (0-140). What is the most likely diagnosis? A) Acute pancreatitis. B) Ascending cholangitis. C) Hepatitis.

D) Perforated peptic ulcer.

E) Cholecystitis.

Answer → B.

Acute "Ascending" Cholangitis

- ***Charcot's Triad*** **(frj)** →

  Fever, **Right upper quadrant pain**, **Jaundice**.

  ± **HL** (**H**ypOtension and **L**eucocytosis).

  Sometime, the stem may mention high bilirubin instead of jaundice.

- ***Investigations*** → Ultrasound and Blood cultures.

Q) Why not acute pancreatitis?

√ Although amylase is elevated here, but the triad (especially **right upper quadrant pain**) is more towards ascending cholangitis. In acute pancreatitis, the pain is usually **mid-epigastric** or in the **left upper quadrant**.

√ Also, in acute pancreatitis, the **amylase elevation** is usually more than **X3** of the upper normal limit.

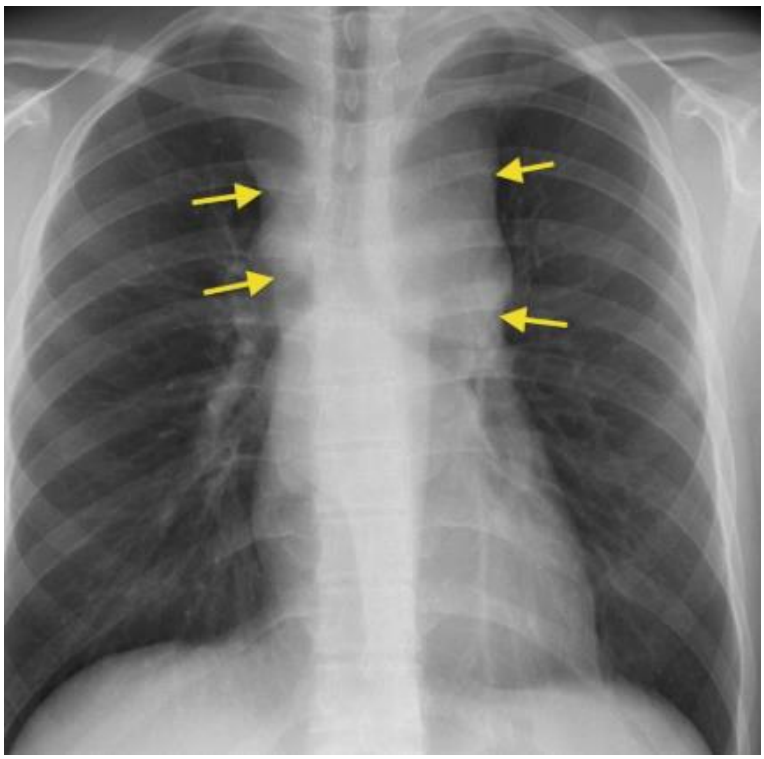
Note*: both ascending cholangitis and acute pancreatitis can occur as complications after ERCP.*

Key 57

Scenario

A patient who has undergone an endoscopy for chronic dyspepsia yesterday returns to the hospital today complaining of chest pain and SOB. The pain is worse at the epigastric area and radiates to the interscapular region of the back. The abdomen is soft, lax, and non-tender. He is tachypnic, tachycardic, mildly feverish with normal hypertension.

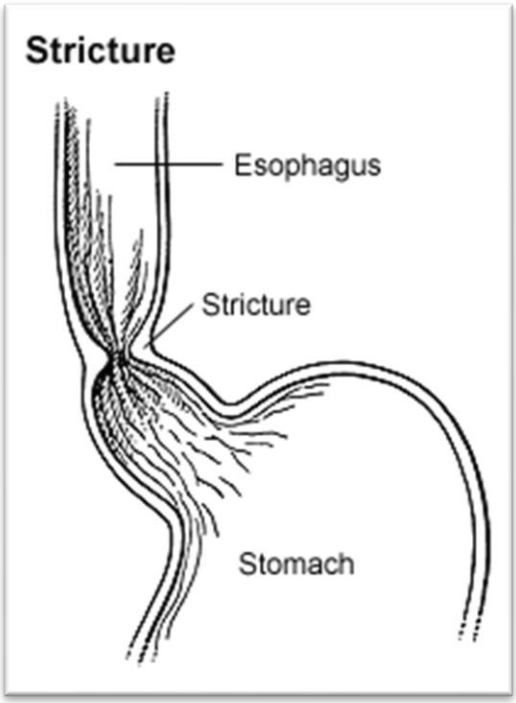
X-ray is performed and shows the following result:



- The likely diagnosis is → **Mediastinitis**. (likely posterior)

- There could be **oesophageal perforation** during the endoscopy performed recently that has led to **mediastinitis**.

	• The X-ray provided shows → **Mediastinal Widening**. • The prominent symptom of mediastinitis is **Chest Pain.** o **Anterior mediastinitis** → pain mainly in the **subcostal area**. o **Posterior mediastinitis** → pain mainly in the **epigastric region** and radiates to the **interscapular region** of the back. • **Treatment → Antibiotics + repairment of the perforation.** • **Notes:** o Normal BP excludes vascular injury (e.g., traumatic aortic injury). o Clear lung fields exclude aspiration pneumonia and pneumothorax.
Key 58	Slowly **progressive Dysphagia** in a young adult. No Weight loss. Hb is normal. Hx of taking **H2-blockers** (e.g. Ranitidine) for **retrosternal discomfort** (**GORD**) for long period → think of a benign stricture (e.g. **peptic stricture**).



√ The history of taking H2-blockers indicates that the patient has a long complaint of GERD.

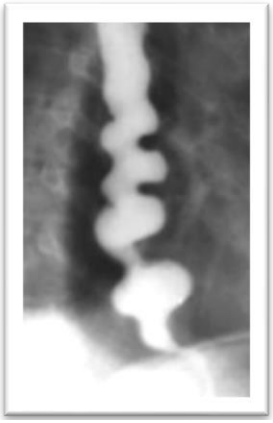
√ **The reflux is erosive → can cause scar and thus → stricture.**

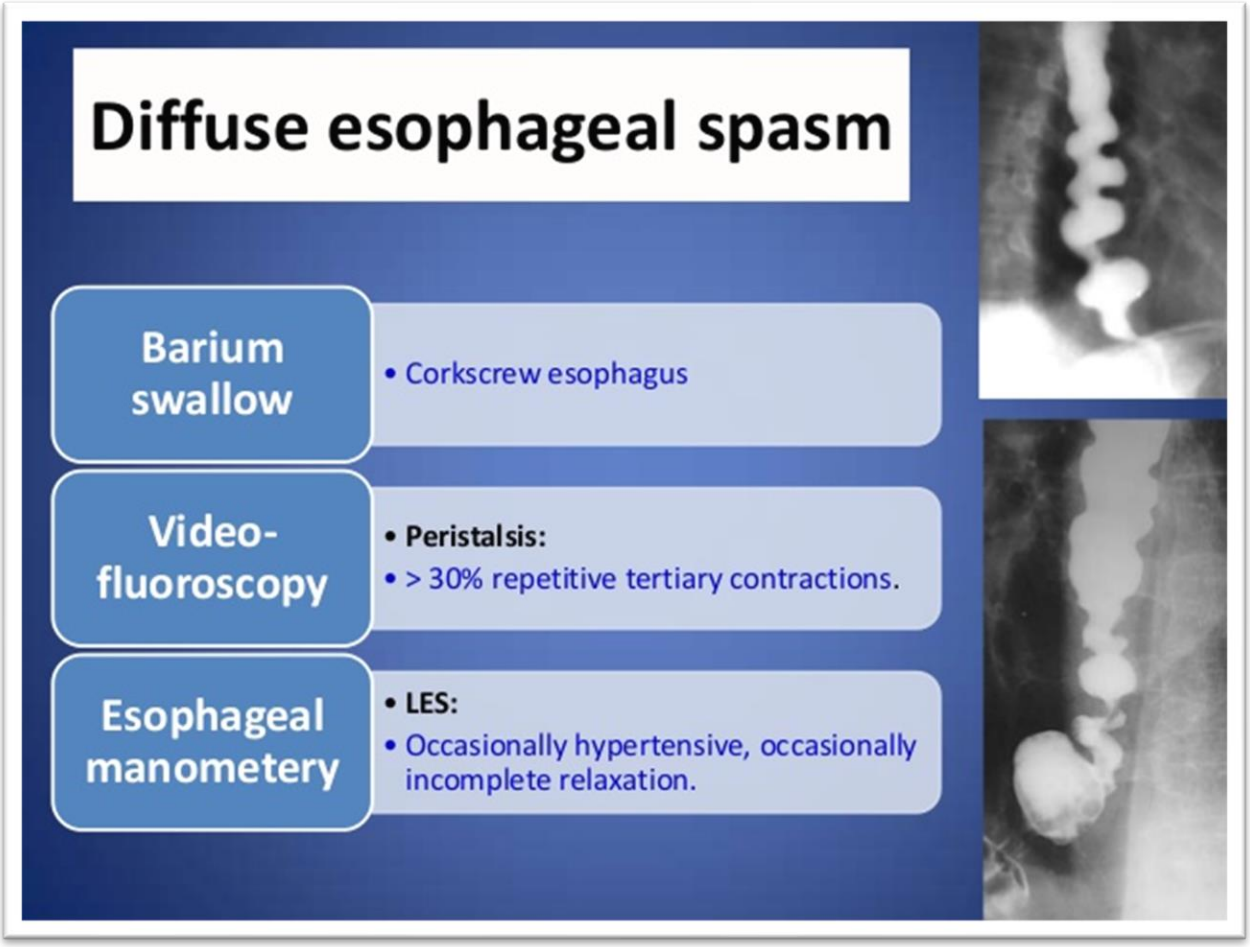
Key 59

- **TTF-1** (Thyroid Transcription Factor 1) is a marker for
  → 1ry **pulmonary adenocarcinoma**.

- Metastasis from **Lung** to **Liver** can occur **Mainly** via
  → (**Haematogenous** route).

- Metastasis form LUNG to LIVER → **Hematogenous**

	Lung → heart → aorta → celiac trunk → common hepatic artery → **liver**
Key 60	**Oesophageal Spasm (Diffuse Oesophageal Spasm) (DES)** o **Intermittent** and **Unpredictable** **SEVERE Chest Pain.** o "***Retrosternal Pain similar to MI but with no relation to exertion***" √ Also, ECG and cardiac enzymes are normal. **+ Dysphagia**. o Aggravated by **cold drinks**, **large amount of water**.  ◙ **Investigations:** o **Barium meal** → **Corkscrew appearance of oesophagus** (at spasm time). o **The most accurate and best test** → **Manometry** ***√ diagnostic*** → **High intensity disorganised contractions**. ◙ **Management:** → **CCB "Calcium Channel Blockers"** (e.g., **N**ifedipine) and **N**itrates **√**. (N and N) *Remember, Spasm takes 2 Ns: Nifedipine (CCB) and Nitrates*.



Example:

42 Y/O male attends the A&E with **severe crushing retrosternal pain** associated with dysphagia. It is noticed especially when he drinks **cold water**. He is given sublingual nitroglycerin and the pain subsides. There is occasional regurgitation of undigested food. ECG shows sinus rhythm. Cardiac enzymes are normal. Vitals are normal.

- The likely diagnosis → **Oesophageal spasm**.
- The best diagnostic investigation → **Manometry**.

Key 61	Chronic **bloody** diarrhea + biopsy shows **crypt abscess** → **Ulcerative Colitis** • **Points towards Crohn's disease** o Endoscopy → **Skip lesions**, **Transmural** (deep Ulcers), **Cobblestone** appearance o Histology → **Granuloma**. **↑ Goblet cells**. o Examination → Abdominal Pain or Mass on the **RIGHT** iliac fossa. o **Diarrhea** "Usually **Non-bloody**". o **Weight loss** is more common. o **Fistulae, perianal fistulas.** • **Points towards Ulcerative Colitis** o Barium enema → **Loss of haustration**, **drain pipe** appearance. o Histology → **Crypt Abscesses**. **(↓) Goblet Cells**. o Abdominal pain on **LEFT** lower quadrant. o **Bloody Diarrhea** is more common. o **Primary Sclerosing Cholangitis** is more common.

	Aphthous oral ulcers occur in both CD and UC, however, slightly more common in **CD**. --- **Histology on biopsy**: ◙ **CD**: granuloma, high goblet cells. ◙ **UC**: no granuloma, there is crypt abscess, low goblet cells
Key 62	**Crohn's Disease Treatment** 1st line → **Prednisolone**. 2nd line → **Budesonide**. 3rd line → 5-ASA (e.g. **Mesalazine**) ***If prednisolone and Budesonide are not given in the options, pick Mesalazine.*** --- ◙ **Treatment of Inflammatory Bowel Disease in Short:** • **Crohn's Disease** → Oral **Prednisolone** (**1st line for remission**). Or **budesonide**

	If both are Not given in the options, pick **Mesalazine (as it is the 2nd line)** Mnemonic: **C**rohn's → **C**orticosteroids (prednisolone) 1st line. • **Ulcerative Colitis** → **5-ASA (Mesalazine)**. (**1st line to induce remission**) • **severe UC exacerbation (Toxic Megacolon)** → Pick **IV Hydrocortisone**.
Key 63	**Severe** shooting recurrent **episodes** of rectal pain that are brief, transient and self-limiting **without** any abnormalities seen on Sigmoidoscopy. → **Proctalgia Fugax** Proctalgia fugax is anal pain that doesn't have a specific cause. This pain is usually caused by intense muscle spasms in or around the canal of the anus. It's similar to another type of anal pain called Levator ani syndrome. The pain is slightly different in Levator ani syndrome, and may last days instead of minutes.

Key 64	• **Liver biopsy** shows large amounts of iron pigment within hepatocytes → **Haemochromatosis**. • **Liver biopsy** shows large amounts of iron pigment within Kupffer cells → **Hemosiderosis**.
Key 65	Left Supraclavicular lump (Virchow's Node) + Weight Loss + Anorexia + Old age → **Gastric Cancer**.
Key 66	A 40-year old male presents with diarrhea, abdominal pain and weight loss for the last 4 months. He has finger clubbing, perianal skin tags. Colonoscopy shows Transmural Granulomatous inflammation involving the ileocecal junction. The likely diagnosis → **Crohn's disease**. √ Non bloody diarrhea. Weight Loss. Perianal Skin tags → more common in CD than in UC. √ The transmural ileocecal (right side) inflammation → CD. **For your knowledge:**

Common Causes for finger Clubbing:

Causes of Clubbing

C → Cyanotic Heart Disease (e.g. Fallot's tetralogy, TGA)
Cystic Fibrosis
L → Lung cancer.
Lung Abscess.
U → Ulcerative Colitis and Crohn's Disease.
B → Bronchiectasis
B → Benign Mesothelioma.
I → Infective "Bacterial" Endocarditis.
Idiopathic Pulmonary Fibrosis.
N → Neurogenic tumors.
G → GIT disease.

Secondary Causes of Clubbing

Cardiac	Respiratory	Gastrointestinal	Other
• Infective endocarditis • Congenital heart disease • Atrial myxoma • Sickle cell disease	• Bronchial carcinoma • Bronchiectasis • Empyema • Cystic fibrosis • Interstitial lung disease • Lung abscess • Mesothelioma • Sarcoidosis	• Inflammatory bowel disease • GI lymphoma • Celiac disease • Hepatic cirrhosis	• Thyroid acropatchy (Graves') • Familial or congenital • Malignancy • Pregnancy

Key 67	An elderly patient with Hx of smoking attends complaining of inability to swallow both solids and liquids for 10 months. Barium swallow shows oesophageal dilatation with a smooth narrowing at the distal end of the oesophagus. The likely diagnosis → **Achalasia** ***The old age and Hx of smoking given here are distractors.*** **Achalasia**: o Inability to relax the lower oesophageal sphincter. O on manometry ⟶ Increased lower oesophageal resting pressure. O Barium swallow ⟶ bird beak appearance (Dilated Oesophagus that tapers -Narrows- Towards the distal end of the oesophagus) o Rx ⟶ Dilatation of the lower oesophageal sphincter.
Key 68	An elderly woman residing the nursing home complains of 5 days constipation. She is on analgesics for her osteoarthritis and back pain. Rectal examination reveals hard impacted stools. The immediate management → **Phosphate Enema**.

However, if young, healthy, no comorbidities, try Glycerol suppositories first. "important".

Management of Constipation

- Impacted stool → **Phosphate Enema**.

However, if young, healthy, no comorbidities, try Glycerol suppositories first. "important".

- **Hard stool** but not impacted → **Stool softeners**.

- Constipation with **soft stools**

→ **High fibre diet** → **Senna = (stimulant laxatives)** (**1st line**),

→ **Lactulose or Macrogol** (ie, **osmotic laxatives**) (2nd line) in general.

- **Pregnancy with constipation** →

√ First line → Ispaghula husk (bulk-forming laxative).

√ Second line → Lactulose (osmotic laxative).

√ Third line → Senna (stimulant laxative).

Ie, **lactulose** is preferred over **senna** in pregnant women.

- This elderly woman has primarily prolonged **constipation** that has led to **fecal impaction**. The food does not pass but the water passes and flow around the blockage; thus, she presented with diarrhea. This is called

→ **Overflow (Spurious) diarrhea**.

	• The cause of her constipation might be due to **opioids** (Analgesics) as they reduce the intestinal peristalsis → Fecal impaction and constipation. • Fecal impaction is also a common complication of constipation in those residing "the **nursing home**" and those who are **bedridden**.
Key 69	• **Acute cholecystitis is fairly common with pregnancy**. This is because the pregnancy changes the composition of the bile **(+)** it slows the emptying of the gallbladder → gallstone formation. • ALP is **normally** elevated during pregnancy and early postpartum. **Scenario with analysis** A woman who has just delivered her baby 3 days ago presents with severe epigastric pain, nausea and chills. Her BP was normal during the pregnancy and is still normal. Her temperature is 37.3°C. Urinalysis shows no protein. ALT 600 (high) ALP 600 (high) Bilirubin 25 (high) Hb 102 (low, but normal for postpartum period) WBC 14 (high) Platelet 330 (normal) INR 1 (normal: 0.8 – 1.2)

→ **Acute Cholecystitis**

- The presenting symptoms + The recent Hx of pregnancy make the acute cholecystitis more likely.

- **Why not HELLP syndrome?**

We can see that platelet is normal and Hb is normal for postpartum period.

(In **HELLP** → **H**emolysis (Low Hb), **E**levated **L**iver enzymes, **L**ow **P**latelets).

- **Why Not Acute fatty liver of pregnancy (AFLP)?**

  Platelet count is **normal** + there is **no hypoglycemia nor ↑ ammonia**.

  Remember that:

- **HELLP** Syndrome → **H**emolysis, **E**levated **L**iver enzymes, **L**ow **P**latelets.
- AFLP → **ELLP** (without **H**emolysis) + (↓) Glucose ± (↑) Ammonia

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	• **Why not pre-eclampsia?** There is **no hypertension nor protein in the urine**. • **Why not Obstetric cholestasis?** In obstetric cholestasis, there would be **severe pruritus** (due to ↑ bile salts) **◙ Low Hb in pregnancy, when can we call it Anemia?** √ In 1st trimester → if the Hb **< 11** g/dL. √ In 2nd Trimester → If Hb **< 10.5** g/dL. √ In 3rd Trimester → If Hb **< 10.5** g/dL. √ **Post-Partum** → If Hb **< 10** g/dL.
Key 70	Intermittent diarrhea + abdominal bloating + Fatigue Think → **Celiac Disease**.
Key 71	Severe central abdominal pain radiates to the back and relieves when bending forwards + Vomiting

	→ **Acute Pancreatitis** (classical presentation). ***What is the next step in the management?*** → initially Supportive **IV fluid "Fluid resuscitation"** + **Analgesics** + **Nutritional support**
Key 72	**Old age** + **Weight Loss** + Chronic Abdominal pain, Bloating and **Diarrhea** or **Constipation** + **Anemia** → **Perform Colonoscopy** (suspected colon cancer)
Key 73	**Scenario** 55 Y/O male. Hx of worsening, intermittent dysphagia. Occasional Regurgitation of food. Recurrent respiratory infections. No weight loss. → **Achalasia**.

	The word "Regurgitation" → **Achalasia** or **Pharyngeal pouch**. In pharyngeal pouch, there will be other specific features (e.g., ***Halitosis, gurgling sound on drinking,*** a ***sensation of a lump in the throat, neck bulge)***. ***N.B.*** *If there was a Hx of Weight Loss, we would think of Oesophageal cancer.* ***N.B.*** *The recurrent chest infections mentioned are mainly due to aspiration pneumonia due to the regurgitation seen in achalasia.*
Key 74	**Chronic Pancreatitis** • The major cause is **Alcohol** (80% of the cases). Others: autoimmune, smoking. • **Epigastric pain that radiates to the back**. • The pain is **episodic (comes and go); (Remitting and relapsing).** • The pain is aggravated by **eating**. • **Weight loss** → due to the ***fear of eating*** "as eating usually exacerbates epigastric pain" + due to the ***malabsorption***. • **Steatorrhea** (Pale, offensive and loose stool that is difficult to flush) → This occurs due to malabsorption. Remember that the healthy pancreas secretes **lipase enzyme** that digest the fat and facilitates its absorption.

- Later on, → **DM** and **Jaundice**.

√ It is different from Acute pancreatitis in the chronic nature of the disease. i.e. the symptoms of the epigastric pain come and go over a long period of time.

√ Also, the serum Amylase and Lipase are **not** usually very elevated in chronic pancreatitis.

- **Investigations:**
  - Initial → U/S abdomen and X-ray abdomen.
  - Abdominal X-ray → **Diffuse abdominal calcifications**.
  - **The gold standard** → **Spiral CT Abdomen with Contrast**

    → shows pancreatic **calcifications**.
  - A useful investigation in chronic pancreatitis

    → **FECAL elastase** "it would be low" and also **FECAL chymotrypsin**.

    "fecal, not serum"!

- **Treatment:**
  - Analgesics.
  - Pancreatic enzyme supplements.

	o Fat soluble vitamins.
Key 75	**Remember that** • In **Ulcerative colitis** → the histology shows **↓ Goblet cells**. • In the **Crohn's disease** → there is **↑** in the **Goblet cells** + **Granulomas**
Key 76	**Remember** In **Pernicious Anemia**, Vitamin B12 Deficiency develops. The treatment in this case is **Intramuscular Hydroxocobalamin**. **We cannot give Oral Vitamin B12 Supplements. Why?** Pernicious Anemia → Autoimmune Atrophic Gastritis → ***Loss of gastric intrinsic factors*** that are required for Vit B12 absorption. Therefore, **Oral tablets will not be absorbed** due to the loss of the gastric intrinsic factors. **What if the cause of Vit. B12 Deficiency is dietary (in Vegans)?** → **Oral Vitamin B12 supplement can be given**. ***N.B. Cobalamin = Vitamin B12***

Key 77	**Alcoholism** + **Haematemesis (± Melena)** + Signs of Chronic Liver Disease (e.g. **Ascites**, **Spider Naevi**). → **Oesophageal Varices** (Due to chronic liver disease caused by alcohol and led to PHT -Portal Hypertension- and thus oesophageal varices). N.B. **Do not rush into Mallory-Weiss Tear**! It is just an oesophageal **tear** that develops due to repetitive vomiting and retching seen in alcoholics especially after a night full of alcohol intake. **There won't be associated signs of liver disease** such as ascites or spider naevi. **How To manage Acute Oesophageal Varices Bleeding?** • **ABC** (Including IV fluid resuscitation) • **Terlipressin** and **prophylactic antibiotics** should be given to patients at presentation (i.e. before endoscopy) • **Endoscopy** → **Band ligation** "first line" should be used for oesophageal varices and injections of N-butyl-2-cyanoacrylate for patients with gastric varices. N.B. If Band ligation is not available → **Sclerotherapy** as first line. • **Transjugular intrahepatic portosystemic shunts** (**TIPS**) should be offered if bleeding from varices is not controlled with the above measures.

	N.B. We do not give PPI (Proton Pump Inhibitors) if the suspected cause of the Oesophageal bleeding is Varices. **Otherwise, if the cause is peptic ulcer for instance, we can give IV PPI.**
Key 78	• Diarrhea → > 3 watery or loose stool per day. • Acute Diarrhea → < 14 days. • Chronic diarrhea → > 14 days. **A patient with diarrhea sometimes bloody for 7 days (Still Acute) + Abdominal pain. What to do next?** Order → **Stool culture, microscopy and sensitivity**. • It is still too early to decide on whether this is a case of Ulcerative colitis or no. It has been there for 7 days only (still acute). • We begin with **stool microscopy, culture and sensitivity** **to exclude any infectious disease** caused by organisms such as E. Coli, Shigella, Salmonella, Campylobacter pylori, Giardia and so on. • Then, accordingly, we may proceed to **Colonoscopy + Biopsy**.
Key 79	**Barret's oesophagus**

Under the prolonged hydrochloric acid reflux to the oesophagus (in those having GORD) → the lower oesophagus undergoes "**Metaplasia**" which means that the epithelial lining the mucosa of the lower oesophagus will change from squamous to columnar epithelium.

[**Squamous** epithelium turns to **Columnar** epithelium **with goblet cells**]

Therefore, the change that is expected to be seen on the histology of the lower third of oesophagus in patients with Barret's oesophagus is:

→ **Columnar Metaplasia**.

This is a precancerous condition as it can develop to oesophageal **Adenocarcinoma** of the lower 1/3 of the oesophagus.

N.B.

Achalasia → **SCC** of the **upper 2/3** of the oesophagus.

Barret → **Adenocarcinoma** of the **lower 1/3** of the oesophagus.

(Adenocarcinoma of the oesophagus is Common in **GERD** and **Barret's oesoph.)**.

Key 80	Sudden onset of **epigastric pain that radiates to back** and **relieved by sitting and bending forwards** + **Vomiting** + Hx of **alcohol** or **Gallstones** or ERCP or Trauma ± fever → Classical for **Acute Pancreatitis**.
Key 81	**Gilbert's Syndrome** • Autosomal recessive **Unconjugated** (indirect) hyperbilirubinemia. • Due to decreased activity of **UGT-1 enzyme** which conjugates the bilirubin with the glucuronic acid. • Can be **precipitated** by (**infection**), (**stress**), (**fasting**) …etc. (episodic). • All **liver function tests are normal** except for a **"mild" ↑ bilirubin**. • Usually, it is ***asymptomatic*** but can present with jaundice only ± Hx of a recent infection or episodic yellowing of eyes. • Although it is unconjugated hyperbilirubinemia, the **reticulocyte** count is **normal** because the reason is not hemolysis. • **Urine dipstick** is also **normal**. • ***Comparison Point:*** In **Dubin Johnson's syndrome**, the raised bilirubin is the **Conjugated** (Direct) bilirubin; thus, **urine dipstick is abnormal** and shows hyperbilirubinemia.

In short:

Isolated jaundice (↑ bilirubin) with all other tests being normal → Think: Gilbert's syndrome.

Scenario

A 20 Y/O male

Presents with yellow sclera and skin

Hx of URTI

Urine dipstick is normal

Bilirubin is 40 (mildly raised)

ALT, AST, ALP, Albumin, Hb, Reticulocytes are **normal**

The likely diagnosis → **Gilbert's syndrome**

- **Why not G6PD?**

  In G6PD, there is hemolysis; thus, ↑ reticulocytes.

- **Why not Acute hepatitis?**

  In acute hepatitis, tremendously ↑ ALT and AST.

- **Why not Dubin Johnson's Syndrome?**

  In DJS, urine dipstick will show bilirubin. (Conjugated).

Key 82	Severe pain on defecation + tender, reddish blue swelling **near** the anus **The likely Dx** → **Perianal hematoma** or (External thrombosed hematoma) **In Haemorrhoids, they are not painful unless infected or thrombosed.** **Also, there is usually a sort of bleeding Hx such as blood on tissue when wiping. In Addition, haemorrhoids are within the anus, not near it or around it.**
Key 83	**Scenario** 40 Y/O male with Hx of **EPISODIC** "remitting and relapsing" upper abdominal pain has visited the hospital several times for this pain in the last several months. The **serum amylase** was gradually increasing on each visit. U/S and Endoscopy were done but found to be normal. The abdomen is soft but mildly tender at the epigastrium. **What is the likely diagnosis**? → **Chronic pancreatitis**. **What is the commonest cause**? → **Excessive alcohol intake**. **What is the best next investigation**? → **CT pancreas with contrast**. o A useful investigation in chronic pancreatitis → **FECAL elastase** "it would be low" and also **FECAL chymotrypsin**. "Fecal, not serum"!

Key 84	Diarrhea, bloody sometimes, pain and tenderness in lower right quadrant, weight loss, oral ulcers, smoker → **Crohn's disease** ***Although bloody diarrhea is not common in CD compared to UC, it still can occur. All other given features are more common in CD than in UC.***
Key 85	**Constipation management [Scenarios]** **[Scenario 1]: An elderly man with prostate cancer and bone metastasis complains of passing stools only once every four days. He drinks adequate fluids and describes his stool as soft. What is the management?** This patient is obviously taking **opioids** to control his pain due to bone metastasis. A common side effect of opioids is **Constipation**. • Since there is **no impacted stool** → **No phosphate enema**. • Since there is **no hard stool** → **no stool softeners**. • Low residue diet = Low fibre diet → Absolutely wrong as ***we should give high fibre diet*** in constipation. • We are left with (**Stimulant laxatives** such as **Senna**) and (**Osmotic laxatives** such as **Lactulose** and **Macrogol**). **The Order of Constipation Interventions is as follows:**

1) High fibre (residue) diet. (for long-term, not for the acute constipation).

2) Senna (Stimulant Laxatives).

3) Lactulose or Macrogol or Polyethylene glycol (Oral Osmotic Laxatives).

4) Add a prokinetic agent (such as domperidone, metoclopramide, erythromycin).

5) Dantron.

6) Seek specialist advice.

According to this order, the answer is → **Senna** (**Stimulant Laxatives**).

N.B. Senna is tried before osmotic laxatives eg, lactulose in general. However, in pregnancy, we use lactulose as Senna might be harmful.

[Scenario 2]: A 74-year-old woman with a history of hypothyroidism and a chronic use of opioids for knee osteoarthritis presents to the ER complaining of nausea, abdominal bloating, and an inability to pass stools or gas for several days. Despite taking senna tablets, her constipation has worsened significantly over the past few weeks. On examination, her abdomen is distended with minimal tenderness, her rectum is empty on digital rectal examination. What is the most appropriate initial treatment?

This patient is on **opioids** to control his pain due to knee osteoarthritis. A common side effect of opioids is **Constipation**.

- Since there is **no impacted stool (empty rectum)** → **No phosphate enema**.

- Since there is **no hard stool** → **no stool softeners**.
- High fibre diet → will **not** be helpful in such an **acute** case. (used for long-term constipation).
- She is already on **Stimulant laxatives** eg, **Senna**) but no improvement.
- We are left with **Oral Osmotic laxatives** such as **Lactulose**, **Macrogol**, **Polyethylene glycol**) → The stool impaction is more proximal "above in the colon" as the rectum is empty. Oral osmotic laxatives can draw water into the intestine and therefore stimulate bowel movements.

The Order of Constipation Interventions is as follows:

1) High fibre (residue) diet. (for long-term, not for the acute constipation).

2) Senna (Stimulant Laxatives).

3) Lactulose or Macrogol or Polyethylene glycol (Oral Osmotic Laxatives).

4) Add a prokinetic agent (such as domperidone, metoclopramide, erythromycin).

5) Dantron.

6) Seek specialist advice.

According to this order, the answer is → (**Oral osmotic laxatives**).

Very Important Note:

If there was **impacted stools** (large fecal mass) in either the **rectum** or even the **sigmoid colon**, the initial treatment step is → Phosphate enema.

Key 86

The sites of the main absorption of:

- **Iron** → **Duodenum**.
- **Folic Acid (Folate)** → **Duodenum** and **Jejunum**.
- **Vit B12** → **Terminal** (**Distal**) **Ileum**.
- **Bile salts → Terminal** (**Distal**) **Ileum.**
- **The majority of nutrients → Jejunum.**

To make it easy, memorise the following:

Iron → duodenum.

Folate → jejunum (and duodenum).

Vit B12 → Terminal ileum.

Q1) A man with folate deficiency. What is the affected organ (if any)?

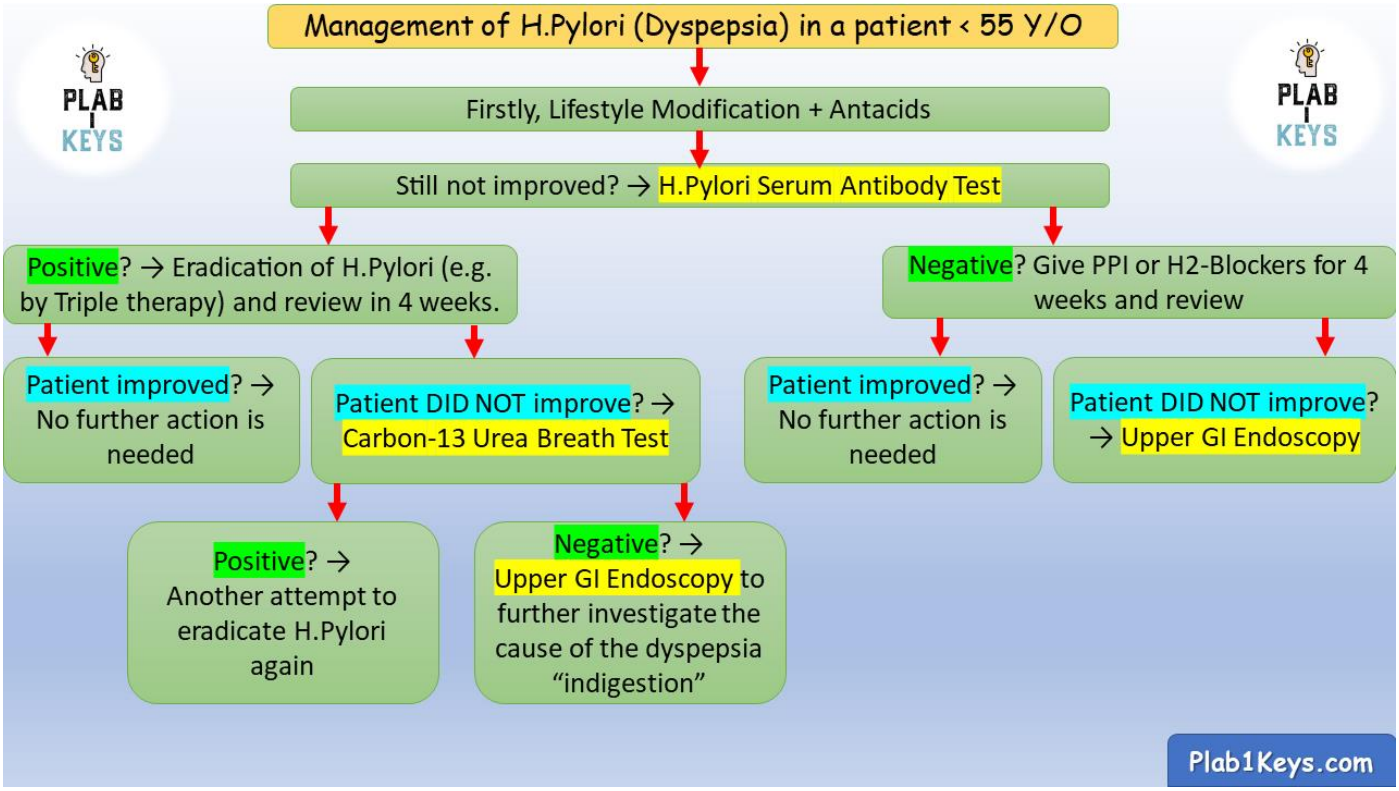
→ Duodenum/ jejunum (proximal small intestine) eg, crohn's, celiac.

Q2) Ileal resection (malabsorption) + Fatigue and Palpitation (due to Anemia) + low hemoglobin and High MCV. What is the deficiency?

→ Vitamin B12 deficiency.

Low Hb and high MCV → megaloblastic (vit. B12 OR Folate deficiency).

Ileal resection? Vitamin B12 is absorbed mainly in ileum, while folate is absorbed mainly in duodenum and jejunum. **So** → Vit. B12 deficiency.

Key 87	40 Y/O male. Intermittent dyspepsia for 4 months. H. Pylori serum antibodies → Negative. Received PPI for 1 month → Not improved. What is next? → **Endoscopy** 
Key 88	**A young woman with Hx of chronic intermittent diarrhea lately becomes bloody with increased frequency of diarrhea and with abdominal pain and**

distension. She is tachycardic, hypotensive, feverish and with high WBCs and CRP and low Hb.

Dx: **Ulcerative Colitis** (**Acute flare – exacerbation**) = **Toxic megacolon**.

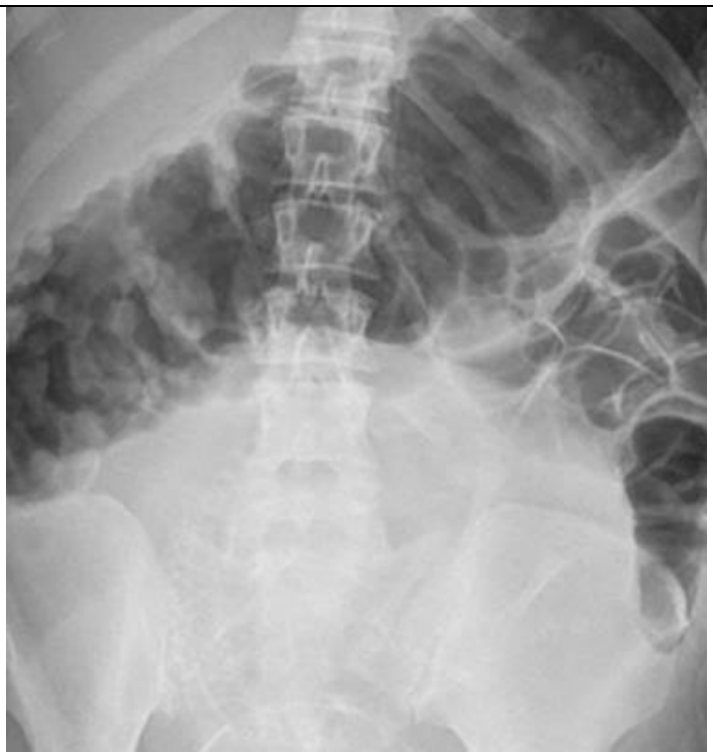
Ix: **Erect abdominal X-ray** → **Mucosal edema** + **TV colon distension**.

Rx: **IV Hydrocortisone**.

When to suspect severe colitis (Acute Exacerbation of UC)?

The rule of **6**, **30**, **90**

- > **6** bowel movements a day + Visible blood in large amounts.
- ESR > **30** (also WBC may be elevated).
- HR > **90** "mildly tachycardic"
- Temperature > 37.8 "mild feverish"
- Anemia (low Hb): (Pallor, Fatigue …etc).

	 **Toxic Megacolon on erect Abdominal X-ray** → ***Dilated colon with thumb printing (Mural Edema) especially in the transverse colon.***
Key 89	An elderly male living in nursing home suffers from constipation. He is agitated and slightly confused. Rectal examination reveals **impacted stool**. What is the management? o Impacted stool → **Phosphate Enema**. o Hard stool but not impacted → Stool softeners. o Constipation with soft stools → High fibre diet → Senna (1st line), Lactulose or Macrogol (2nd line) o Pregnancy with constipation → Lactulose (1st line).
Key 90	**Abdominal Migraine** **(Do not Forget this!)**

	• Seen mainly in **children** aged 5-9 years but can occur in adults. • **No abnormal findings** on examinations and investigations.  • **Characteristics**: o **Episodes** (Paroxysmal) (Attacks) of central or **peri-umbilical severe pain** that lasts for **≥ 1 hour** and **interferes with activities.** o Associated with **episodic headaches** and **2 or more** of the following: **Anorexia**, **Nausea**, **Vomiting**, **Pallor**. • Treatment → **Reassurance**
Key 91	**A patient with celiac disease for 2 years underwent duodenal biopsy that shows lymphomatous infiltrates.** The likely diagnosis → **Lymphoma**. Remember, **T-cell lymphoma** is a rare complication of celiac disease.

	Remember the following conditions that may develop with celiac disease: 1) Iron deficiency Anemia. (The commonest Anemia) 2) Folic Acid Deficiency. (The 2nd Commonest Anemia) 3) Vitamin B12 Deficiency. (The 3rd Commonest Anemia). 4) Osteoporosis. 5) T-Cell Lymphoma (Intestinal Lymphoma). (Rare) 6) Dermatitis Herpetiformis (Skin Rash). 7) DM Type 1.
Key 92	An elderly woman has recently been treated with **broad-spectrum antibiotics** for UTI "urinary tract infection". Now, she develops bloody diarrhea and severe abdominal pain. She is feverish and with high WBCs and CRP. • The Likely Dx → **Pseudomembranous colitis** (**C. Difficile**). ◆ Treatment of choice → **Oral Vancomycin** (1st line) ◆ Sometimes vancomycin would not be given in the choices. So, pick the second line treatment → **Oral Metronidazole**. N.B. for simple UTI, start with low spectrum antibiotic such as ***Trimethoprim*** or ***Nitrofurantoin***.

	This is because the strong "with wide coverage" (broad-spectrum) antibiotics are able to kill the normal gut flora, leaving the clostridium difficile to grow and to attack the GIT, leading to pseudomembranous colitis.
Key 93	Remember that **steatorrhea** can be described in a stem as (**frothy stools that are difficult to flush**). **Steatorrhea** + **Diarrhea** + **Abdominal pain** + **Low Hb** + **Low iron-ferritin-** or **Folic Acid** or **vit B12** √ Think of → **Celiac disease** √ Order → **Tissue Transglutaminase antibodies (IgA).**
Key 94	A woman admitted to a hospital for Pulmonary Embolism management for 3 days. She now develops **epigastric pain**, **diarrhea**, **nausea** but no vomiting. There is no blood in stool. The likely diagnosis → **Gastroenteritis**. • Gastroenteritis is **very common** in admitted patient (in the **hospitals**) particularly due to the abundance of the **Norovirus** in the hospitals.

	• It manifests as an acute onset of: **Diarrhea** + **Abdominal pain** (central or epigastric) ± **Nausea** & **Vomiting**. • The patient should be **isolated** for **48 hours after the diarrhea has resolved**.
Key 95	**An elderly man presents with Back pain, weight loss, Hx of smoking/alcohol, jaundice, High blood glucose. He has palpable liver and gallbladder.** The likely Dx → **Cancer of Pancreas**. **Features of Pancreatic Cancer**: √ Painless Jaundice. √ Hepatomegaly. √ Right upper quadrant mass. √ Weight loss "Wasting" "Cachexia". √ Palpable "non-tender" gallbladder at the right costal margin "Courvoisier's signs" √ Atypical back pain is often seen √ ± Palpable epigastric lump. Palpable Liver. Palpable gallbladder. √ ± Ascites

	Investigations √ Ultrasound has a sensitivity of around 60-90%. √ **High-resolution CT scanning** is the investigation of choice if the diagnosis is suspected. **Management** √ Less than 20% are suitable for surgery at diagnosis. √ A Whipple's resection (pancreaticoduodenectomy) is performed for resectable lesions in the head of pancreas. Side-effects of a Whipple's include dumping syndrome and peptic ulcer disease. √ Adjuvant chemotherapy is usually given following surgery. √ ERCP with stenting is often used for palliation.
Key 96	**A woman admitted to a hospital for Pulmonary Embolism management for 3 days. She now develops abdominal pain, diarrhea, nausea but no vomiting. There is no blood in stool.** The likely diagnosis → **Gastroenteritis**. • Gastroenteritis is **very common** in admitted patient (in the **hospitals**) particularly due to the abundance of the **Norovirus** in the hospitals.

	• It manifests as Acute onset **diarrhea** + **Abdominal pain** (central or epigastric) ± **Nausea & Vomiting**. • The patient should be **isolated** for 48 hours after the diarrhea has resolved.
Key 97	**When can a cook "Food handler" e.g. Chef return to work after an attack of gastroenteritis?** → **48 hours after all symptoms (e.g. Diarrhea, Vomiting) are cleared** In the UK, **Gastroenteritis** patients can return to work after 2 days (**48 hours**) of the **last** episode of symptoms (Diarrhea or Vomiting).
Key 98	**A patient presents with Hx of diarrhea and floating stools. He is anaemic as well. The appropriate investigation?** → **Tissue transglutaminase IgA**. (likely **Celiac Disease**).
Key 99	**An elderly male living in nursing home suffers from constipation. He is agitated and slightly confused. Rectal examination reveals impacted stool. What is the management?**

	- Impacted stool → **Phosphate Enema**. - Hard stool but not impacted → **Stool softeners**. - Constipation with soft stools → High fibre diet → **Senna =** (stimulant laxative) (**1st line**), Lactulose or Macrogol (2nd line) - Pregnancy with constipation → **Lactulose** (1st line).
Key 100	◙ **The gold standard method to diagnose esophogeal carcinoma is** → **Upper GI endoscopy + Biopsy**
Key 101	**A man presents complaining of longstanding regurgitation and progressive dysphagia.** → **Achalasia**. --- "**Regurgitation**" is an alarming word for (**Achalasia**: *Bird Beak Appearance*) and (**Pharyngeal Pouch**).

	However, the latter "pharyngeal Pouch" has other specific features such as ***Halitosis*** "*bad breath smell*", ***Stale food regurgitation*** ± ***Sensation of a lump in the throat*** ± ***Gurgling sounds in the chest when drinking fluids*** ± neck bulge.
Key 102	**A nurse discovers that a patient is heavy alcohol addict; he drinks 30-40 units of alcohol per week. What should be done?** → **Refer to social workers**.
Key 103	**A man presents complaining of regurgitation of undigested food and gurgling sounds on chest when swelling liquid.** → **Pharyngeal pouch** (= Zenker's Diverticulum). **Important Note:** If the dysphagia is progressive and associated with significant weight loss in an old individual, suspect oesophageal carcinoma even if there is gurgling sounds on drinking (it also occurs in cancer).
Key 104	**Gastro-oesophageal reflux disease (GORD)** ◙ **Gastro-oesophageal reflux disease (GORD) may be defined as symptoms of oesophagitis secondary to refluxed gastric contents.**

◙ **Acid reflux is due to poor closure of the lower esophogeal sphincter.**

◙ **Manifestations:**

√ A burning sensation in chest (heartburn), usually after eating, which might be worse at night.

√ Chest pain.

√ Bad breath and bad taste at the back of mouth.

√ Difficulty swallowing.

√ Regurgitation of food or sour liquid.

√ Sensation of a lump in your throat.

◙ **Risk Factors:**

√ Obesity.

√ Pregnancy.

√ Smoking.

√ Large meals "particularly fatty meals".

√ Intake of: coffee, chocolate, alcohol, fat.

√ Medications: Calcium channel blockers, Nitrates, antimuscarinic.

√ Systemic Sclerosis.

√ After treatment of achalasia "that involves dilatation of lower oesophageal sphincter"

√ Hiatus hernia.

◙ **Complications:**

Oesophagitis – ulcers – anaemia – benign strictures – Barrett's oesophagus –

oesophageal carcinoma.

◙ **Management: Proton Pump Inhibitors are the mainstay treatment.**

♦ Endoscopically proven oesophagitis:

√ Full dose proton pump inhibitor (PPI) for 1-2 months.

√ If responsive to Rx → then give low dose treatment as required.

√ If no responsive → then give double-dose PPI for 1 month.

♦ Endoscopically negative reflux disease:

√ Full dose PPI for 1 month.

√ If response → then offer low dose treatment, possibly on an as-required basis, with a limited number of repeat prescriptions.

√ If no response → then offer H2 receptor antagonist (e.g. ranitidine) or prokinetics for one month.

Q) Obese woman with GERD. What is the treatment?

	Give → **Proton pump inhibitor** (PPI).
Key 105	**Liver Cirrhosis with Ascites:** ◙ **Investigation? → Ascitic fluid aspirate analysis: culture, cell count "neutrophil count".** ◙ **Management? → Spironolactone.** ◙ **Other important lines:** **If high neutrophils → IV antibiotics.** **If albumin is low → albumin infusion.**
Key 106	**A young woman with Hx of chronic intermittent diarrhea lately becomes bloody with increased frequency of diarrhea and with abdominal pain and distension. She is tachycardic, hypotensive, feverish and with high WBCs and CRP and low Hb.** **Dx: Ulcerative Colitis (Acute flare – exacerbation) = Toxic megacolon.** **Ix: Erect abdominal X-ray → Mucosal edema + TV colon distension.**

	Rx: **IV Hydrocortisone**.
Key 107	**A man with history of gastrectomy was found to have high MCV.** → **Vitamin B12 Deficiency**. --- **Gastric Resection** → **Malabsorption of Vit B12** → **Macrocytic Anemia** (↑ **MCV**) → **Hypersegmented Neutrophils** on a blood smear. **Vitamin B12 (Cobalamin) Deficiency** **Causes** o **Pernicious Anemia** (the most common cause). Pernicious Anemia → Autoimmune Gastric Atrophy → ***Loss of intrinsic factors*** that are required for Vit B12 absorption. Usually associated with other autoimmune disease e.g. hypothyroidism. o **Total or even partial Gastrectomy** (Impaired Vit B12 Absorption). o **Ileal Resection**. o **Crohn's Disease**.

	○ **Chronic Pancreatitis**. (malabsorption). ○ **Celiac Disease** (malabsorption). ○ **Dietary** (Vegans). Remember that Vit B12 is present in meat, fish and dairy products but not in the vegetables.
Key 108	**Plummer-Vinson syndrome** Triad of: • **Dysphagia** (secondary to **oesophageal webs**) • **Glossitis** • **Iron-deficiency anaemia.** Treatment includes **iron supplementation** and **dilation of the webs**.
Key 109	**Alcoholic Liver Disease**. ♦ Hx of heavy alcohol consumption. ♦ Signs of liver disease/ **cirrhosis**: Ascites, Hematemesis, Jaundice, Hepatomegaly, Spider naevi. ♦ In Alcoholic liver disease:

	Both AST and ALT are elevated; however, AST is more elevated than ALT → **↑ AST:ALT ratio** (e.g., AST:150, ALT: 70). ♦ In Alcoholic liver disease: Gamma Glutamyl Transferase **(GGT) is also increased**. ◙ Stop Alcohol ◙ Spironolactone is given to minimise ascites. ◙ Consider Liver transplant 6 months after alcohol abstinence in late cases.
Key 110	An elderly male living in nursing home suffers from constipation. He is agitated and slightly confused. Rectal examination reveals **impacted stool**. What is the management? o Impacted stool → **Phosphate Enema**. o Hard stool but not impacted → **Stool softeners**. o Constipation with soft stools → **High fibre diet** → **Senna -stimulant laxative**- (1st line), **Lactulose** or **Macrogol** (2nd line) o Pregnancy with constipation → **Lactulose** (1st line).

	A man in home-care on morphine develops difficulty in defecation. His fluid and diet intake are good. His stools are soft. → **Senna** (answer is sometimes: **Stimulant Laxatives**). √ Remember, if soft stools, no impaction, fluid intake is good → Senna (stimulant laxatives) is first line in general. √ Lactulose or macrogol is first line in pregnancy.
Key 111	♠ Remember in Haemochromatosis, the triad: **Hepatomegaly** + **DM** + **Bronze Skin** **± Arthropathy.** ♠ Remember that the "**Liver**" is the most likely organ to get **cancer** in Haemochromatosis (Due to Cirrhosis and iron deposition).
Key 112	◙ **An elderly man with Hodgkin's lymphoma has recently been treated with broad-spectrum antibiotics for UTI. Now, he develops bloody diarrhea and severe abdominal pain.** · The Likely Dx ⟶ **Pseudomembranous colitis** (**Clostridium difficile**). · Treatment of choice ⟶ **Oral Vancomycin** (1st line)

	· Sometimes vancomycin would not be given in the choices. So, pick the second line treatment → **Oral Metronidazole**.
Key 113	◙ vegetarian with megaloblastic anemia (↑ MCV) → **Vitamin B 12 Deficiency**.
Key 114	**Important Oesophageal Cases (DDx)** ◙ A man presents with regurgitation of stale food, halitosis and dysphagia. There is gurgling sounds on drinking: → **Oesophageal pouch** = Zenker's Diverticulum --- ◙ A man presents with regurgitation of stale food and dysphagia. There is gurgling sounds on drinking. The dysphagia is progressive and associated with significant weight loss. He is 66 YO. → **Oesophageal carcinoma** **Important Note:** If the dysphagia is progressive and associated with significant weight loss in an old individual, suspect oesophageal carcinoma even if there is gurgling sounds on drinking (it also occurs in cancer). ---

	◙ A man presents with chest pain, occasional difficulty in swallowing both liquids and solids, recurrent chest infections: The likely Dx ⟶ **Achalasia** - Dysphagia to both liquids and solids. - Recurrent chest infection ⟶ mainly due to Regurgitation (seen in **Achalasia**) ⟶ Aspiration pneumonia.
Key 115	An elderly man presents with left supraclavicular lump and weight loss The likely Dx → **Gastric Cancer**. **Remember**: • Left Supraclavicular Mass → Virchow's Node → Indicative of **Gastric Carcinoma** (Anorexia, Dyspepsia, Weight Loss, Old age). This sign is called → **Troisier's sign**. • Right Supraclavicular Mass → **Oesophageal cancer**, **Lung cancer**, **Hodgkin's Lymphoma**. • **Pancoast Tumour** → A tumour of the **Apex** **of the Lung** (present at the top end of either the left or the right lung). It typically spreads to the nearby

	tissues such as the ***Ribs*** and the ***Vertebrae***. Most Pancoast tumours are Non-small cell lung cancer.
Key 116	**A 58 YO man presents complaining of chronic gastric reflux, dysphagia and chest pain. Initially, there was difficulty in swallowing solid food, but recently, there becomes dysphagia to soft food as well. Barium swallow is done and shows irregular narrowing of the middle third of the thoracic oesophagus with proximal shouldering.** **The likely Dx is → Oesophageal Carcinoma.** **The diagnostic modality → Upper GI Endoscopy and Biopsy.**
Key 117	[**Traveller's Diarrhea**] √ The main cause for traveller's diarrhoea (in general) is → **E. coli**. √ However, other organisms relating to travel can also cause diarrhoea, **depending on the presenting features and the country of travel**. **For example:** • Traveller's diarrhoea that is usually of a short period and self-limited (especially Hx of travel to Africa) → **E. coli**.

• Hx of travel to Europe, WATERY (Non-bloody) diarrhoea, Weight Loss (If chronic), abdominal pain and bloating (Symptoms for > 10 Days) → **Giardia**.

• Hx of travel → Prodrome: HIGH Fever, Headache, Myalgia → **Followed by BLOODY Diarrhea → Campylobacter jejuni**

• Another important cause for traveller's diarrhoea is **Salmonella**, "presents the same as Campylobacter jejuni"

√ Salmonella first line → **Ciprofloxacin**. (A question in Plab 1 September 2019 exam).

√ Campylobacter first line → erythromycin or clarithromycin ▌2nd → **Ciprofloxacin**

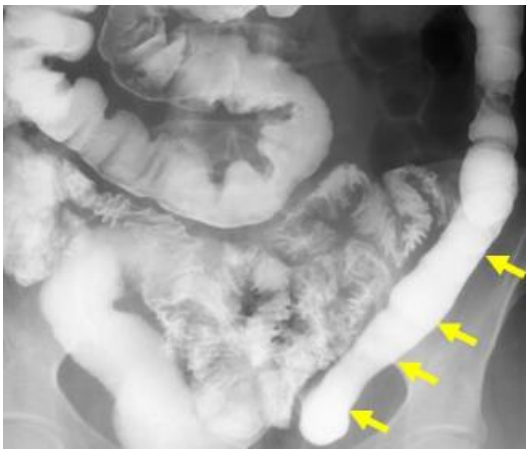
N.B. Campylobacter jejuni is Gram Negative Curved Bacilli (Rods).

(A question in Plab 1 November 2019 exam).

◙ The traveller's diarrhoea can be caused by different organisms, depending on the features and the country of travel.

Key 118	**20yr old with recurrent diarrhoea and abdominal pain on the left side for 6 months, she did a biopsy which showed granulomas and inflammation. What is the most likely diagnosis?** a) UC b) **CD (Crohn's Disease)** c) Tb abdomen d) Colorectal Ca √ Do not get tricked by the presence of the pain in the left side. Remember that in CD, the presence of mass is usually in right side "at ileocecal junction". However, **CD can affect any part of the GIT from mouth to anus**! √ **Granuloma** is a common histological finding in **CD**. √ Also, in UC, the diarrhea is usually bloody.
Key 119	**Barium enema of ulcerative colitis would show →** **Loss of haustration.** **Notes:** • **Crohn's disease → colonoscopy → Cobble stone appearance, Deep ulcers, Skip lesions.** • **Crohn's disease → Small Bowel Enema → Kantor's string sign, thorn ulcers and fistulae.**

- **Ulcerative colitis → Barium enema → Loss of haustral markings.**



Loss of Haustra in UC.

Key 120

Oesophageal Spasm (Diffuse Oesophageal Spasm) (DES)

- **Intermittent** and **Unpredictable** **SEVERE Chest Pain.**
- "***Retrosternal Pain similar to MI but with no relation to exertion***" √ Also, ECG and cardiac enzymes are normal.

+ Dysphagia.

- Aggravated by **cold drinks**. √√
- **Barium meal**

→ **Corkscrew appearance of the oesophagus** (at the spasm time).

- **The most accurate test** → Manometric studies

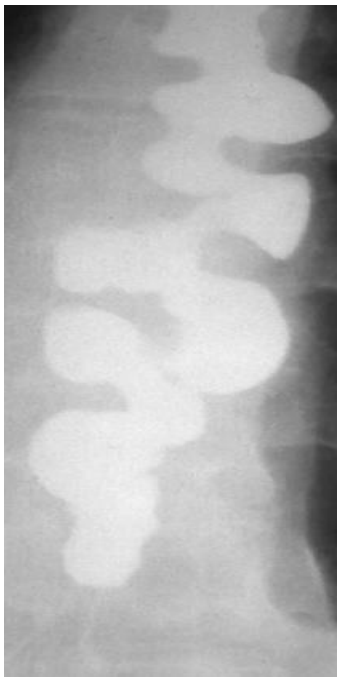
  → **High intensity disorganised contractions**.

- **Rx**

  → **CCB "Calcium Channel Blockers"**

  (e.g., **N**ifedipine) and **N**itrates √. (N and N)

  Remember, Spasm takes 2 Ns: Nifedipine (CCB) and Nitrates.



Corkscrew oesophagus in DES (Diffuse Oesophageal Spasm) by barium meal.

Mnemonic for DES

C-C-C-C

Corkscrew – **C**hest pain – **C**CB – **C**old liquids.

Notes:

- ***Oesophageal spasm → Corkscrew oesophagus.***
- ***Achalasia → Bird beak appearance of the oesophagus.***

Key 121	**A patient with 3 episodes of hematemesis and epigastric pain. He takes NSAIDs for his Rheumatoid Arthritis.** • **What do we suspect?** → **Peptic Ulcer** (caused by the prolonged use of NSAIDs). • **What should be done**? → **Endoscopy**. **Also, part of the management: *IV fluid*, *Analgesics*, *Antiemetics and IV PPI*.** • **What if the endoscopy is not available at the moment?** → **IV Proton Pump Inhibitors PPI.** • **When do we give IV Antibiotics?** → As a **prophylaxis** in the cases of Upper GI bleeding due to **Oesophageal Varices** OR **Perforated Peptic Ulcer**.
Key 122	**A question about Barret's Oesophagus. What kind of histological change is seen?** A. **Metaplasia**

B. Hyperplasia

C. Dysplasia

D. Carcinoma in-situ

Barret's Oesophagus

Under the prolonged hydrochloric acid reflux to the oesophagus (in those having GORD) → the **lower oesophagus** undergoes "**Metaplasia**" which means that the epithelium lining the mucosa of the lower oesophagus will change from **squamous** to **columnar** epithelium.

[**Squamous** epithelium turns to **Columnar** epithelium **with goblet cells**]

S → **C**

Shampoo for **C**hildren

Squamous → **C**olumnar

Therefore, the change that is expected to be seen on the histology of the lower third of oesophagus in patients with Barret's oesophagus is:

→ **Columnar Metaplasia**.

This is a precancerous condition as it can develop to oesophageal **Adenocarcinoma** of the lower 1/3 of the oesophagus.

N.B.

	Achalasia → **SCC** of the **upper 2/3** of the oesophagus. **Barret** → **Adenocarcinoma** of the **lower 1/3** of the oesophagus **(Adenocarcinoma** of the oesophagus is Common in **GERD** and **Barret's oesophagus)**.
Key 123	**Elderly man 62 years of age, Alcoholic, presents with difficulty in swallowing solid food and weight loss of 3 months. What is the Most likely diagnosis?** A. **Esophogeal Carcinoma** B. Achalasia C. C. Pharyngeal pouch **Factors That would raise our suspicion to oesophageal cancer:** *Old age* ± *weight loss* ± Hx of *smoking, Alcohol* ± *Anemia* ± ***Increasing*** *"Progressive" dysphagia* ***to solids then to Fluids*** ± *Hx of Barret's Oesophagus* ± *Hx of GORD* • **The diagnostic Investigation** → **Endoscopy + Biopsy**. **Notes on Oesophageal Carcinoma** o The most common type → **Adenocarcinoma**. o Smoking → Associated more with **SCC**.

	○ Barret's Oesophagus → a precursor of **Adenocarcinoma**. ○ Achalasia → chronic inflammation → more risk for **SCC**. ○ Upper 2/3 of the oesophagus → **SCC**. ○ Lower 1/3 of the oesophagus → **Adenocarcinoma**.
Key 124	**57 yr old presents with difficulty to swallow solids and weight loss of 5 months. What is the investigation that will lead to a diagnosis?** a. Barium swallow b. **Upper GI endoscopy** c. Manometry d. Ct scan **Risk Factors That would raise our suspicion to oesophageal cancer:** *Old age* ± *weight loss* ± Hx of *smoking, Alcohol* ± *Anemia* ± ***Increasing*** *"Progressive" dysphagia to* ***solids*** *then to Fluids* ± *Hx of Barret's Oesophagus* ± *Hx of GORD* • **The diagnostic Investigation** → Endoscopy + Biopsy.
Key 125	**6 months post stroke patient with intermittent dysphagia. He has lost weight and has chest infection. What is the best way to administer nutrition?**

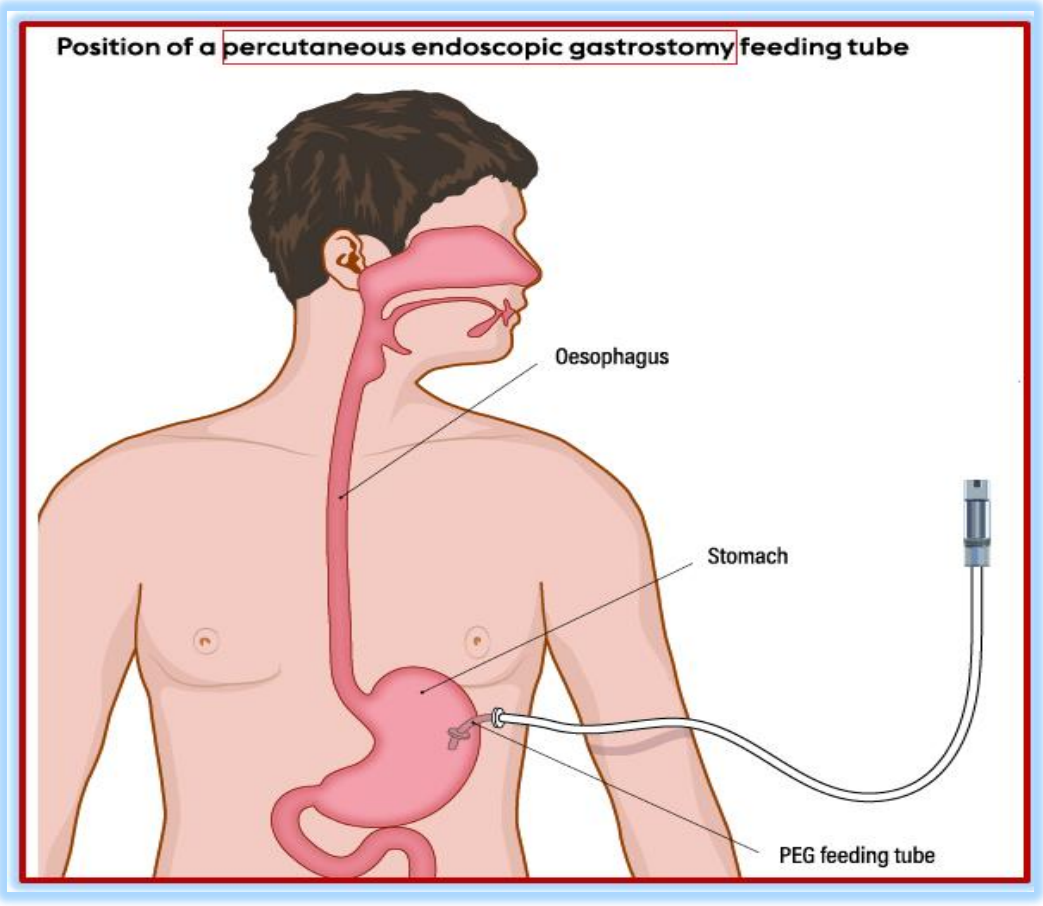
a) Parenteral nutrition

b) **Percutaneous gastrostomy**

c) NG tube

d) Feeding jejunostomy

Stroke patients sometimes have difficulty in swallowing which can lead to aspiration pneumonia and thus recurrent chest infection. This is seen in this patient who also has started to lose weight. He needs an alternative **long-term feeding** option.



	In patients with **Oesophageal cancer with liver metastasis** → No surgery. (End-stage oesophageal cancer is inoperable). **So, how to relieve the symptom of Severe Dysphagia?** → **Oesophageal stenting** √ Insert a stent into the oesophagus, it will expand and open the obstruction (Fast and Effective).
Key 126	**A patient with cirrhosis has progressive abdominal distension, spider nevi, shifting dullness, generalized abdominal pain, rebound tenderness, absent bowel sounds; and fever.** ◙ **Investigation →** **Neutrophil count from ascitic fluid aspirate.** ◙ **Initial management →** **Start antibiotics immediately** √ This is a likely case of [**Spontaneous bacterial peritonitis**] which is a common complication of ascites.

	√ The organism gains access to the peritoneum usually by hematogenous spread. √ **The best initial test** is to **aspirate 10-20 ml of the ascitic fluid and to perform cell count**: (neutrophils > 250 cells / µL) necessitates the start of the antibiotics immediately. √ **The most accurate test** is → **culture of the ascitic fluid aspirate** "but it takes days". **Important**: Rx of **ascites** secondary to Cirrhosis (alcohol abuse, ascites, spider naevi) without fever, tenderness etc (without spontaneous bacterial peritonitis): → **Spironolactone** (Potassium-sparing diuretics).
Key 127	**After ERCP, a man developed right hypochondriac pain with fever, elevated bilirubin and raised amylase. What is the likely diagnosis?** A. **Ascending cholangitis** B. Acute pancreatitis C. Cholangitis D. Acute cholecystitis **Confirmed by** → Ultrasound abdomen + Blood cultures.

Acute "Ascending" Cholangitis:

- **Charcot's Triad** (**FRJ**)

  → **Fever**, **Right upper quadrant pain**, **Jaundice (raised bilirubin)**.

  ± **HL** (**H**ypOtension and **L**eucocytosis).

- ***Investigations*** → Ultrasound and Blood cultures.

- ***Management:***

Fluid resuscitation

Broad-spectrum intravenous antibiotics

Correct any coagulopathy

Early ERCP

Another similar question with a different answer (be careful)!

After ERCP, a man developed epigastric pain that radiates to the back and eases on leaning forward. He also has some nausea, vomiting, jaundice "elevated bilirubin" and raised amylase. What is the likely diagnosis?

A. Ascending cholangitis

	B. **Acute pancreatitis** C. Cholangitis D. Acute cholecystitis ◙ **Confirmed by** → High serum lipase and amylase + CT pancreas. ◙ **Management?** → initially Supportive **IV fluid "Fluid resuscitation"** + **Analgesics** + **Nutritional support**
Key 128	**Important Differentials of Liver Disease** o **1ry Biliary Cirrhosis** → **Middle-aged female, Pruritus, Jaundice, ↑ ALP, associated with Sjogren's Syndrome.** **Investigation: Anti-Mitochondrial Antibodies.** o **1ry Sclerosing Cholangitis** → **the same but the association is usually IBD (mainly Ulcerative colitis).**

Investigation: ERCP

- **Autoimmune hepatitis → Early-middle aged female, abnormal ALT and AST, Normal or mildly elevated ALP ± 2ry Amenorrhea ± another autoimmune disease (e.g. hypothyroidism, vitiligo, rheumatoid arthritis, celiac, pernicious anemia)**

- **Alcoholic Liver Disease → Hx of heavy alcohol consumption. Signs of liver disease/ cirrhosis: Ascites, Hematemesis, Jaundice, Hepatomegaly, Spider naevi. Both AST and ALT are elevated; however, AST is more elevated than ALT: ↑ AST:ALT ratio (e.g. AST:150, ALT: 70). Gamma Glutamyl Transferase (GGT) is also increased.**

- **Drug-induced hepatitis → Co-amoxiclav (amoxicillin + clavulanic acid), flucloxacillin, can cause hepatitis especially in people with deteriorated liver functions such as chronic alcoholics. Clavulanic acid is highly toxic to liver. If the patient already has risk factors for hepatic impairment such as chronic alcoholism, hepatic excretion of clavulanic acid will be impaired, leading to cholestasis (Jaundice, Dark Urine, Pruritis) ''high bilirubin'' and drug-induced hepatitis (incredibly high ALT, AST, ALP).**

  N.B. drugs that can cause hepatic cholestasis:

  Co-amoxiclav, flucloxacillin, steroids, Sulphonylureas…etc.

Key 129

DDx of Dysphagia

The table below gives characteristic exam question features for conditions causing dysphagia.

Remember that **new-onset dysphagia** is a red flag symptom that requires **urgent endoscopy**, regardless of age or other symptoms. (**New guidelines**).

Causes	Notes
Oesophageal cancer	Dysphagia may be associated with weight loss, anorexia or vomiting during eating. Past history may include Barrett's oesophagus, GORD, excessive smoking or alcohol use.
Oe'ophagitis	There may be a history of heartburn, odynophagia but no weight loss and systemically well.
Oesophageal candidiasis	There may be a history of HIV or other risk factors such as steroid inhaler use "↓ immunity". Presents with dysphagia + odynophagia "burning, pain during swallowing".
Achalasia	Dysphagia of both liquids and solids from the start. Heartburn. Regurgitation of food - may lead to cough, aspiration pneumonia, chest pain, etc. –
Pharyngeal pouch	More common in older men. Represents a posteromedial herniation between thyropharyngeus and cricopharyngeus muscles Usually not seen but if large then a midline lump in the neck that

		gurgles on palpation. Typical symptoms are dysphagia, regurgitation, aspiration and chronic cough. Halitosis and gurgling sound when drinking may occasionally be seen.
	Diffuse Oesophageal Spasm	o Intermittent and Unpredictable SEVERE Chest Pain. o "Retrosternal Pain similar to MI but with no relation to exertion" √ Also, ECG and cardiac enzymes are normal. + Dysphagia. o Aggravated by cold drinks. o Barium meal → Corkscrew appearance of the oesophagus (at the spasm time). o The most accurate test → Manometric studies → High intensity disorganised contractions. o Rx → CCB "Calcium Channel Blockers" (e.g. Nifedipine) and Nitrates √. (N and N)
	Systemic sclerosis	Other features of **CREST** syndrome may be present, namely **C**alcinosis, **R**aynaud's phenomenon, o**E**sophageal dysmotility, **S**clerodactyly, **T**elan'iectasia. As well as oesophageal dysmotility, the lower oesophageal sphincter (LES) pressure is decreased. This contrasts to achalasia where the LES pressure is increased
	Myasthenia gravis	Other symptoms may include extraocular muscle weakness or ptosis. Dysphagia with liquids as well as solids.

	Globus hystericus	There may be a history of anxiety. Symptoms are often intermittent and relieved by swallowing. Usually painless - the presence of pain should warrant further investigation –or organic causes.

Key 130

♦ **Chest pain that radiates to the back [or to the interscapular region]**

+

♦ **Widened mediastinum on Chest X-ray**

What is the likely Dx?

√ **If there is Hx of recent endoscopy** & **the vitals are relatively stable**

Think → **Mediastinitis** "due to oesophageal perforation".

√ **If No Hx of recent endoscopy** & **the vitals are worsening** (esp. ↓ BP)

Think → **Thoracic artery dissection/ rupture**.

Key 131	**A 40 YO female known case of rheumatoid arthritis on celecoxib (a NSAID) is brough to the A&E complaining of 3 episodes of vomiting blood. Her BP is 75/55. What is the NEXT step?** → **Give IV fluids**. √ As the patient is **haemodynamically unstable** (the SBP <90), the next step is to administer IV fluids for circulation stability. You administer IV fluids while preparing for blood transfusion (cross-match and blood group). √ If the patient deteriorated while on IV fluids and no time for cross matching and blood grouping, O negative blood may be given. √ This hematemesis is likely due to gastric ulcer 2ry to the prolonged use of NSAIDs for her rheumatoid arthritis. √ After stabilisation, endoscopy would need to be done. √ IV proton pump inhibitors are useful in the case of gastric ulcer; however, they can be given after the endoscopy.
Key 132	**Hepatitis A** Hepatitis A is typically a benign, self-limiting disease, with a serious outcome being very rare.

◙ **Overview:**

√ incubation period: 2-4 weeks

√ RNA picornavirus

√ transmission is by faecal-oral spread, often in institutions

√ **Shellfish** are reservoirs of hepatitis A. Therefore, if hepatitis features appear after having seafood meal, think of hepatitis A as a differential.

Request → IgM antibodies to hepatitis A virus. **√ important**

Also, if IgG is positive, we need to do IgM to see if it is acute "still active".

√ doesn't cause chronic disease

√ management is supportive: fluids,'anti-emetics… .

√ Hepatitis is a notifiable disease.

◙ **Features**

√ flu-like prodrome

√ abdominal pain: typically, right upper quadrant

√ tender hepatomegaly

√ jaundice

√ cholestatic liver function tests

◙ **Complications**

complications are rare and there is no increased risk of hepatocellular cancer

◙ **Immunisation**

an effective vaccine is available

after the initial dose, a booster dose should be given 6-12 months later

◙ **Investigations:**

√ Both ALT and AST are high "However, ALT is much higher".

√ high bilirubin → jaundice, dark urine and pale stools can be seen.

√ high alkaline phosphatase.

√ Anti-hepatitis A IgM antibodies → detected around the time the symptoms appear → indicating acute hepatitis A.

√ Anti-hepatitis A IgG antibodies → detected soon after IgM and remains detectable for life.

• The presence of IgM with/without IgG indicates acute infection.

• The presence of isolated IgG indicates that the patient had been infected in the past.

Key 133

Hepatitis B

◙ Hepatitis B is a double-stranded DNA hepadnavirus and is spread through exposure to infected blood or body fluids, including vertical transmission from mother to child. The incubation period is 6-20 weeks.

◙ The features of hepatitis B include fever, jaundice and elevated liver transaminases.

◙ Complications of hepatitis B infection

chronic hepatitis (5-10%). 'Ground-glass' hepatocytes may be seen on light microscopy

f'lminant live' failure (1%)

hepatocellular carcinoma

glomerulonephritis

polyarteritis nodosa

cryoglobulinaemia

◙ **Immunisation against hepatitis B:**

√ children born in the UK are now vaccinated as part of the routine immunisation schedule. This is given at 2, 3 and 4 months of age.

√ at risk groups who should be vaccinated include: healthcare workers, intravenous drug users, sex workers, close family contacts of an individual

with hepatitis B, individuals receiving blood transfusions regularly, chronic kidney disease patients who may soon require renal replacement therapy, prisoners, chronic liver disease patients

◙ **Management of hepatitis B**

√ pegylated interferon-alpha used to be the only treatment available. It reduces viral replication in up to 30% of chronic carriers. A better response is predicted by being female, < 50 years old, low HBV DNA levels, non-Asian, HIV negative, high degree of inflammation on liver biopsy

√ whilst NICE still advocate the use of pegylated interferon first-line other antiviral medications are increasingly used with an aim to suppress viral replication (not in a dissimilar way to treating HIV patients)

√ examples include tenofovir, entecavir and telbivudine (a synthetic thymidine nucleoside analogue)

Hepatitis B Serology (important points to memorise)

• HB**sAg** (+ve) → during **acute** and **chronic** infection "The **first** marker that **becomes abnormal** after acquiring Hepatitis B infection".

• HB**sAg** (+ve) and HB**eAg** (+ve) → Highly infectious "Active viral replication" **(***eager to spread***)**

√ What if antibodies against this (**e**) develop?

→ Anti-**HBe** → **Indicates response to treatment.**

• Anti-**HBs** (+ve) → post vaccination/ recovery and immunity against HBV.

(*Vaccine comes from Harvard Business School "HBs"*)

• Which Antibodies will be +ve at the onset of symptoms and will remain +ve even after treatment "**Indicates Past or ongoing infection**"?

→ The **core** antibody "Anti-**HBc**"

Note → s=surface | e=envelope | c=core | Ag=Antigen | Anti=Antibodies

In summary (a must-know points):

◙ The **first marker** to become abnormal → **HBsAg**. (**Acute/Chronic** infection)

◙ Indicates **high infectivity** → **HBeAg**.

◙ Indicates **recent vacIt isination** → **Anti-HBs**

◙ Indicates **past infection** → **Anti-HBc**

Additional:

IgM anti-HBc → Recent **acute** infection.

	HBV **DNA** → Infectivity (Active viral replication)
Key 134	**Hepatitis C** ◙ Hepatitis C is likely to become a significant public health problem in the UK in the next decade. It is thought around 200,000 people are chronically infected with the virus. At risk groups include intravenous drug users and patients who received a blood transfusion prior to 1991 (e.g. haemophiliacs). ◙ **Pathophysiology:** √ hepatitis C is a RNA flavivirus √ incubation period: 6-9 weeks ◙ **Transmission:** **√** the risk of transmission during a needle stick injury is about 2% **√** the vertical transmission rate from mother to child is about 6%. The risk is higher if there is coexistent HIV **√** breastfeeding is **not** contraindicated in mothers with hepatitis C **√** the risk of transmitting the virus during sexual intercourse is probably less than 5%

	√ there is no current vaccine for hepatitis C ◙ After exposure to the hepatitis C virus, only around 30% of patients will develop features such as: √ a transient rise in serum aminotransferases / jaundice √ fatigue √ arthralgia ◙ **Investigations:** √ HCV RNA is the investigation of choice to diagnose acute infection. √ whilst patients will eventually develop anti-HCV antibodies, it should be remembered that patients who spontaneously clear the virus will continue to have anti-HCV antibodies ◙ **Outcome** around 15-45% of patients will clear the virus after an acute infection (depending on their age and underlying health) and hence the majority (55-85%) will develop chronic hepatitis C ◙ ◙ **Chronic hepatitis C** ◙ ◙

• Chronic hepatitis C may be defined as the persistence of HCV RNA in the blood for 6 months.

• **Potential complications of chronic hepatitis C:**

√ rheumatological problems: arthralgia, arthritis

√ eye problems: Sjogren's syndrome

√ cirrhosis (5-20% of those with chronic disease’

√ hepatocellular cancer

√ cryoglobulinaemia: typically type II (mixed monoclonal and polyclonal)

√ porphyria cutanea tarda (PCT): it is increasingly recognised that PCT may develop in patients with hepatitis C, especially if there are other factors such as alcohol abuse

√ membranoproliferative glomerulonephritis

◙ **Management of chronic infection:**

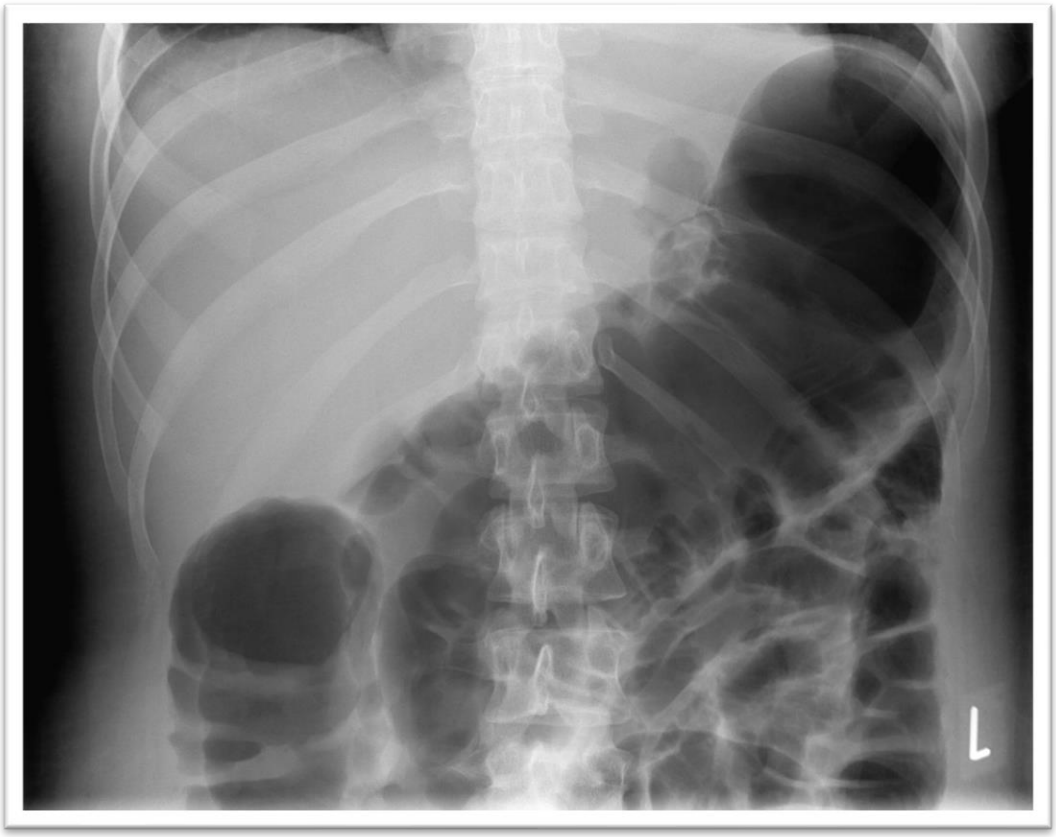
√ treatment depends on the viral genotype - this should be tested prior to treatment.

√ the management–of hepatitis C has advanced rapidly in recent years resulting in clearance rates of around 95%. Interferon based treatments are no longer recommended.

	√ the aim of treatment is sustained virological response (SVR), defined as undetectable serum HCV RNA six months after the end of therapy. √ currently a combination of protease inhibitors (e.g. daclatasvir + sofosbuvir or sofosbuvir + simeprevir) with or without ribavirin are used. ◙ **Complications of treatment:** √ ribavirin - side-effects: haemolytic anaemia, cough. Women should not – ecome pregnant within 6 months of stopping ribavirin as it is teratogenic. √ interferon alpha - side-effects: flu-like symptoms, depression, fatigue, leuk–penia, thrombocytopenia.
Key 135	**Hepatitis D** ◙ Hepatitis D is a single stranded RNA virus that is transmitted parenterally. It is an incomplete RNA virus that requires hepatitis B surface antigen to complete its replication and transmission cycle. ◙ It is transmitted in a similar fashion to hepatitis B (exchange of bodily fluids) and patients may be infected with hepatitis B and hepatitis D at the same time. ◙ Hepatitis D terminology:

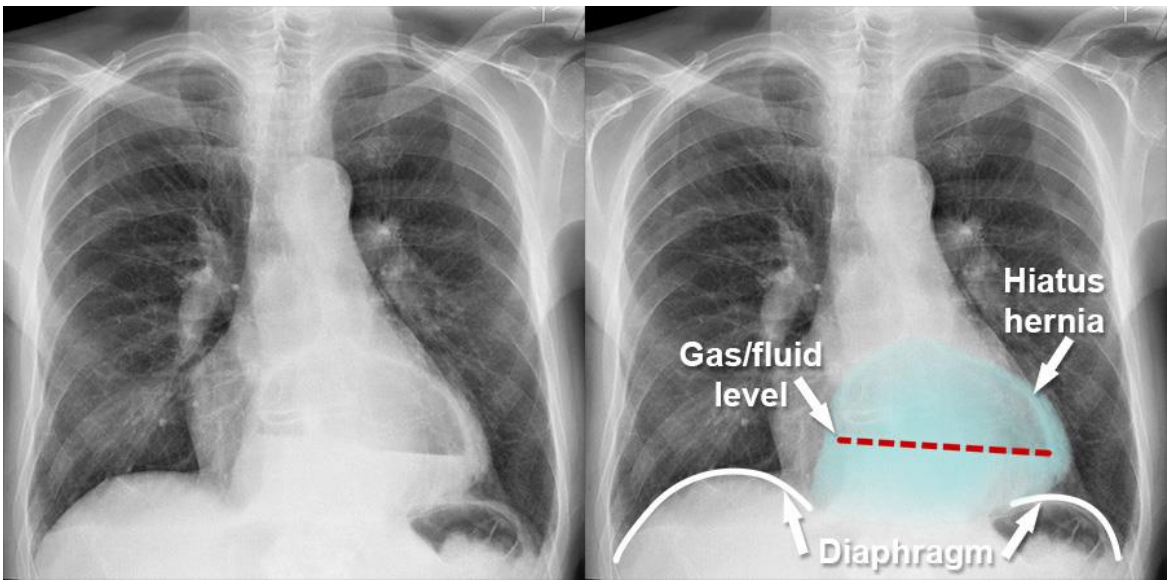
	√ Co-infection: Hepatitis B and Hepatitis D infection at the same time. √ Superinfection: a Hepatitis B surface antigen positive patient subsequently develops hepatitis D infection. Superinfection is associated with high risk of fulminant hepatitis, chronic hepatitis status and cirrhosis. ◙ Diagnosis is made via reverse polymerase chain reaction of hepatitis D RNA. ◙ Interferon is currently used as treatment, but with a poor evidence base.
Key 136	**Hepatitis E** √ RNA hepevirus. √ spread by the faecal-oral route. √ incubation period: 3-8 weeks. √ common in Central and South-East Asia, North and West Africa, and in Mexico. √ causes a similar disease to hepatitis A, but carries a significant mortality (about 20%) during pregnancy.

	√ does not cause chronic disease or an increased risk of hepatocellular cancer. √ a vaccine is currently in development*, but is not yet in widespread use.
Key 137	Old age Left lower abdominal pain Fever Tender mass at the left iliac fossa An important DDx to consider → Diverticulitis → Diverticular abscess.
Key 138	• GORD → Long-standing heartburn and Dysphagia. • Smoking and Hiatus hernia → exacerbates GORD. • Chronic GORD → can lead to inflammatory changes of the lower oesophagus → Squamous epithelium turns to Columnar → Barret's oesophagus (a premalignant condition with 10% risk to turn to adenocarcinoma).

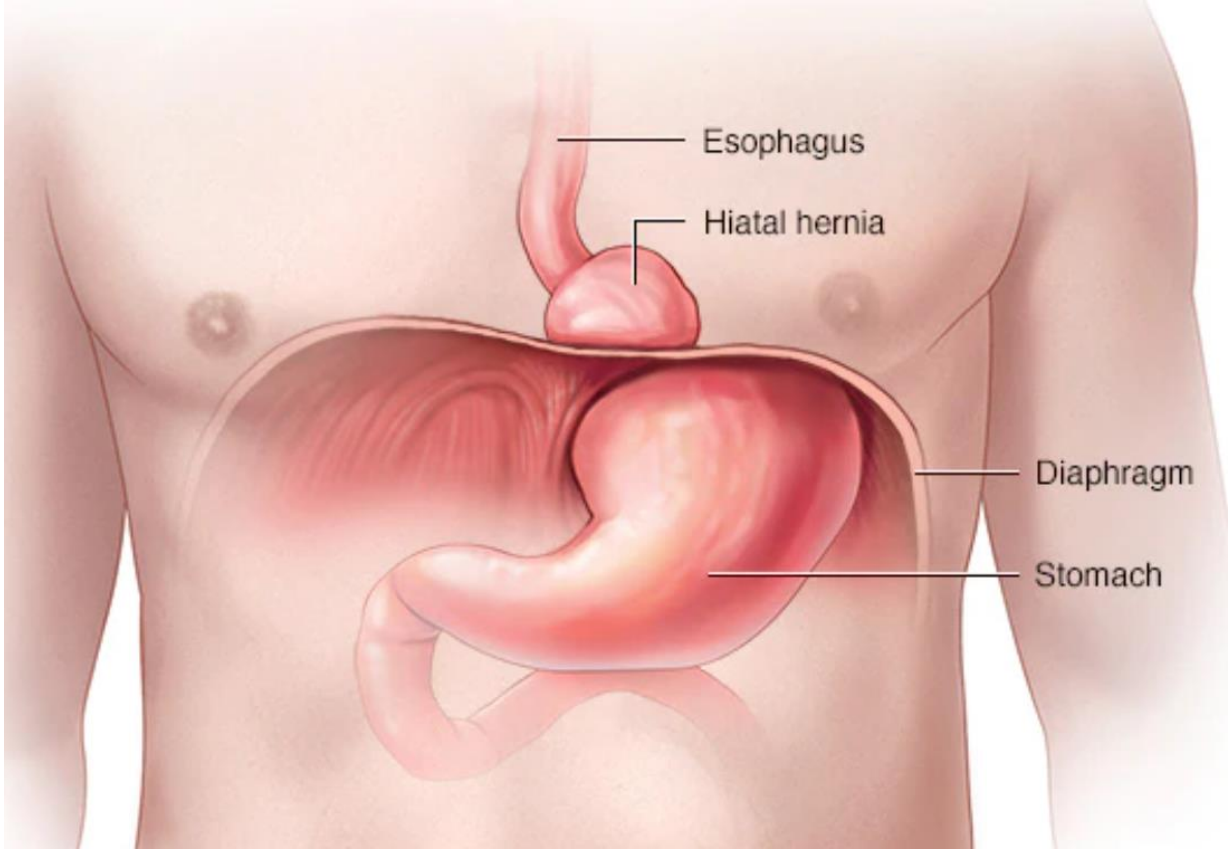
Key 139	 **Toxic megacolon in a patient with a background of ulcerative colitis who presented with shortness of breath, bloody diarrhea, fever and abdominal pain.** This patient needs immediate IV hydrocortisone + IV fluid.
Key 140	**Stress ulcer** (Curling's ulcer) can develop after stress conditions e.g., after surgery, burning, serious infection…etc Give → IV proton pump inhibitors to reduce the risk of bleeding. It can be shifted to oral PPIs after 72 hours.

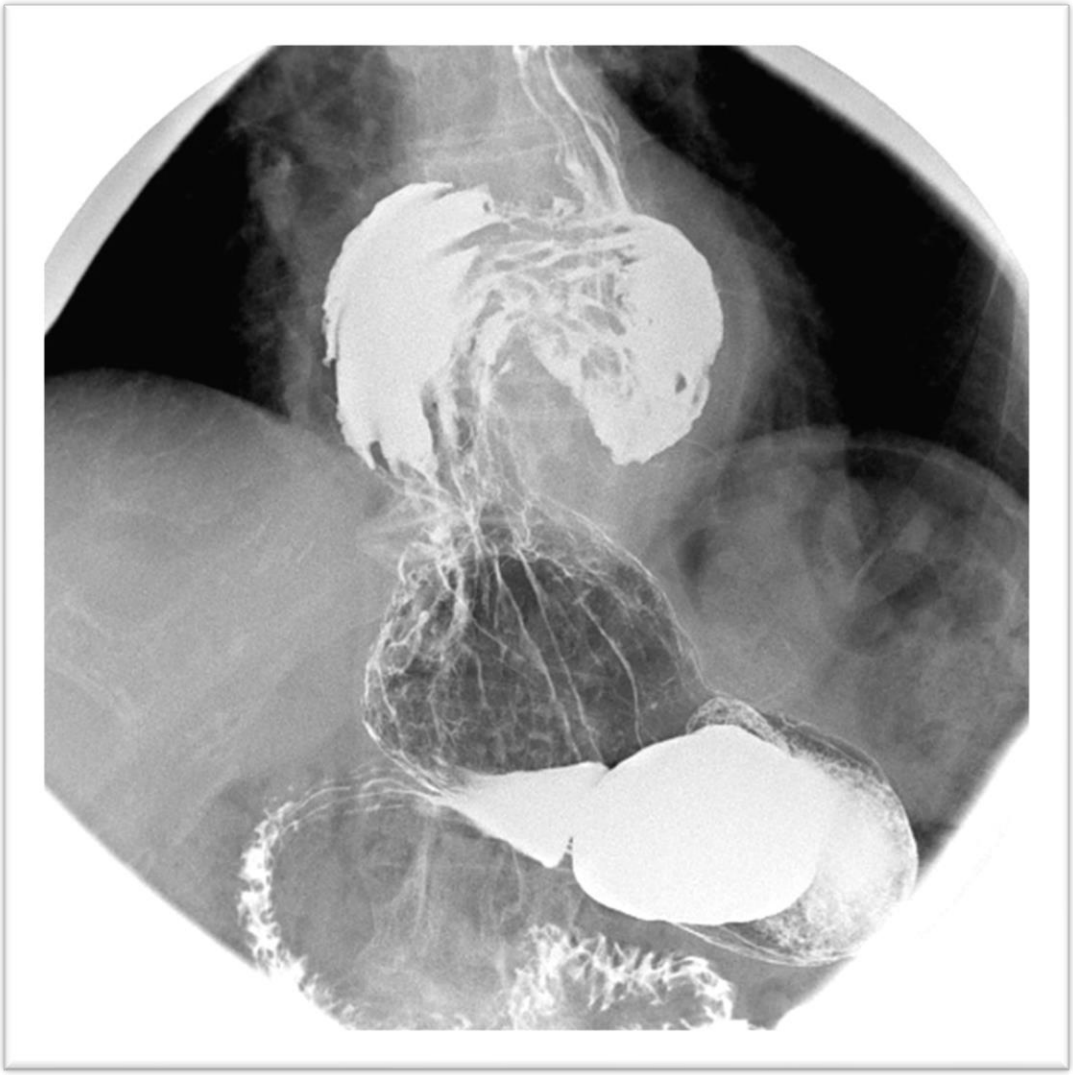
Key 141	**New-onset jaundice + Over 40 YO + Hx of alcohol + abnormal LFTs + Weight loss** → Refer for suspected pancreatic cancer (CT abdomen is needed).
Key 142	**The most definitive (gold standard) investigation that leads to a diagnosis of inflammatory bowel disease (CD, UC) is** → **Colonoscopy.** √ Note that **fecal calprotectin** is useful as it would be raised during active colon inflammation (contrarily, it would be normal in irritable bowel syndrome). However, colonoscopy is the diagnostic approach as it would visualize the colon for the inflammation and ulcers. √ Note that **proctoscopy** is benifical for internal haemorrhoids but would not be that helpful in inflammatory bowel diseases as it would not reach and visualize the entire colon. **Important:** Note that if the question describes a case of a young patient who has features of CD or UC "young, bloody diarrhea" and the question is asking about **the most appropriate Next step**, the answer would be → **Fecal calprotectin**.

	However, if old, and cancer is suspected → **colonoscopy**.
Key 143	**A 56 YO man presents compaining of 3 months of intermittent epigastric pain at night + unintentional weight loss of 5 kg. He drinks about 20 units of alcohol weekly for the past 5 years. O/E, the abdomn is soft, non-tender and no abdominal masses.** **What is the most appropriate investigation?** → **Urgent oesophago-gastro-duodenoscopy (urgent upper endoscopy)**. √ ≥ 55 YO (+) Dyspepsia (+) Weight loss → "urgent" upper GI endoscopy to exclude oesophagel/ gastric cancer. √ This patient has a Hx of heavy alcohol consumption which is a risk factor for oesophageal and gastric cancers and also to a chronic pancreatitis. However, cancer should be investigated urgently.
Key 144	√ **Any patient with dyspepsia who have been taking medications (H2 blockers, PPI) for at least 1 month WITHOUT improvement of symptoms** →**oesophagogastroduodenoscopy (OGD = endoscopy)**. √ **If developed DYSPHAGIA** → **Urgent oesophago-gastro-duodenoscopy (OGD = endoscopy)**.

Key 145	◙ Hx of chronic alcoholism, Ascites (abdominal distension), Hematemesis, Jaundice (high bilirubin), Hepatomegaly, Spider naevi Think → liver cirrhosis. The best diuretic in cirrhosis → Spironolactone.
Key 146	**Burning sensation in chest or retrosternal "especially when bending or lying"** + **Nausea and vomiting**, + **Chest pain**, + **Chest X-ray showing → air-fluid level in a mass behind the heart** ◙ Think → **Hiatus hernia**.  • Hiatus hernia → a herniation of part of the stomach above the diaphragm.

	• There are two types: √ sliding: accounts for 95% of hiatus hernias, the gastroesophageal junction moves above the diaphragm. √ rolling (paraoesophageal): the gastroesophageal junctions remain below the diaphragm but a separate part of the stomach herniates through the oesophageal hiatus • Rx: √ Medical → PPIs. √ Surgery → Laparoscopic fundoplication. "Considered if persistent symptoms e.g., regurgitation, intractable cough, oesophagitis despite PPIs".





This is a **barium swallow** for a 35 YO patient who has been having **persistent heartburn**. It shows → a **sliding hiatus hernia**.

The gastric fundus is displaced above the diaphragm.

Key 147

Irritable bowel syndrome

◙ **The diagnosis of IBS should be considered if the patient has had any of the following for at least 6 months:**

√ Abdominal pain, and/or

√ Bloating, and/or

√ Change in bowel habit

◙ A **positive diagnosis of IBS** should be made if the patient has abdominal pain relieved by defecation or associated with altered bowel frequency ± stool form, in addition to 2 of the following 4 symptoms:

√ Altered stool passage (straining, urgency, incomplete evacuation)

√ Abdominal bloating (more common in women than men), distension, tension or hardness

√ Symptoms made worse by eating, relieved by defecation.

√ Passage of mucus

◙ **Red flag features should be enquired about: (need more Ix for cancer)**

√ Rectal bleeding

√ Unexplained/unintentional weight loss

√ Family history of bowel or ovarian cancer

√ Onset after 60 years of age

◙ **There is no diagnostic test for IBS. However, suggested investigations (for DDx):**

√ FBC, ESR/CRP

√ IgA tissue transglutaminase antibodies → (for coeliac disease).

√ Fecal calprotectin → (for inflammatory bowel disease: UC, CD).

√ Fecal immunochemical test (FIT) → (for colon cancer: generally, > 50 YO).

√ CA-125 → (for ovarian cancer: generally, women > 50 YO).

◙ **IBS can be:**

Constipation predominant or Diarrhea predominant or Mixed.

◙ **Management of IBS:**

√ **Low FODMAP diet**.

FODMAPs or fermentable oligosaccharides, disaccharides, monosaccharides, and polyols are short chain carbohydrates that are poorly absorbed in the small intestine and are prone to absorb water and ferment in the colon.

√ **Antispasmodics**: e.g., mebeverine, peppermint oil.

√ **Laxatives**: e.g., Ispaghula husk "for constipation predominant type of IBS".

√ **Anti-diarrheal**: e.g., Loperamide: "for diarrhea predominant IBS".

◙ Scenario (1):

A 20 YO man has been complaining of abdominal pain for 7 months. On most days, he also has abdominal bloating. The abdominal pain and bloating are worse after eating and they become better after passing stools. He sometimes has mucus in his stool. There is no weight loss or abdominal tenderness.

The most likely Dx → irritable bowel syndrome (IBS).

◙ Scenario (2):

A 33 YO man has been complaining of the passage of loose stool and vague abdominal pain over the past 6 months. His father had gastric cancer. The patient denies any mucous or blood in hist stool. He has no weight loss. On examination, he has generalised abdominal tenderness with no palpable masses.

The most likely Dx → irritable bowel syndrome (IBS).

◙ Sometimes, you would face such vague questions. It would be more obvious that it is IBS if the stem mentions things such as (the pain is relieved when he defecates), or (the patient is very stressed with his new job…etc). These things make IBS easier to pick.

◙ Anyway, IBS also presents with loose stool (diarrhea), and vague abdominal pain.

◙ "Generalised" abdominal tenderness is also seen in IBS due to gases (bloating). Remember, IBS can present with either constipation, or diarrhea or mixed.

◙ Why not inflammatory bowel disease?

√ In **ulcerative colitis**, you would expect more severe symptoms, and mostly blood in stool.

√ In **crohn's disease**, you would expect diarrhea with weight loss and right iliac fossa pain.

◙ Could this case be gastric cancer?

Although there is family history of gastric cancer in this case, it is not likely in this age (<55). In addition, there is no history of dysphagia, abdominal mass, weight loss, or epigastric pain. So, stomach cancer in his father is a risk factor here but not the likely current diagnosis.

◙ What investigations would be needed here?

There is no diagnostic test for IBS. However, it is important to exclude similar DDx such as inflammatory bowel disease (by fecal calprotectin) and celiac disease (by IgA tissue transglutaminase antibodies).

Key 148	A 50 YO woman presents to the A&E for severe tiredness, lethargy, and black tarry stools for the past 3 days. She is a chronic user of naproxen (NSAIDS) for her rheumatoid arthritis. Her blood pressure is 80/45. Her HR is 120. Labs show Hemoglobin of 5.5. The ER team have stabilized the patient by giving IV fluids and 4 units of packed RBCs. Now, BP is 95/55, HR is 100. What shoud be done next? → **Urgent Upper GI Endoscopy**. • This patient likely has bleeding ulcer/s causing the black tarry stool and anemia, likely due to the prolonged use of NSAIDs. • Prolonged use of NSAIDs → Gastric/Peptic ulcers → Bleeding from upper GIT → Black tarry stools + Anemia [eg, fatigue, tachycardia, pallor]. • We need to **hemodynamically stabilize the patient first** and then go for **immediate upper GIT endoscopy** to look for the bleeding source and stop it. • Intravenous PPIs (eg, IV omeprazole) are useful for ulcers. However, NICE guidelines advice that they must **not** be commenced in suspected cases before endoscopy. Endoscopy to confirm the diagnosis comes first.

Key 149	**For upper GIT Endoscopy preparation:** → Fasting for 6 hours before the procedure. (Small amount of clear fluid is acceptable up to 2 hrs before procedure). **Q) A man is scheduled for upper GI endoscopy at 7 a.m. He has consumed a few sweets 4 hrs ago and water 1 hr ago.** → **Re-organise schedule (defer the procedure)**.
Key 150	**Dealing with Paracetamol Overdose:** **Start IV N-Acetyl Cysteine immediately after Paracetamol Overdose (without waiting for the serum paracetamol level) if:** √ **Unknown dose**. √ **Unknown time** (Doubtful time) of ingestion. √ **Staggered dose** (all tablets were not taken at the same hour). √ Presenting **> 8 hours** after ingestion. √ Presenting **Unconscious** or with **Liver tenderness** and **Jaundice**. **If not, then → Measure the paracetamol level 4-hours post ingestion (Not Post-admission).**

Note:

Paracetamol Overdose is treated in the **Medical Ward** not in the Psychiatric ward. Thus, sometimes → "Admit to the medical ward" is the correct answer. However, a referral to **psychiatric team** is usually required after finishing the medical treatment.

◙ If pH is **< 7.3** after 24 hours → Refer for a liver specialist centre. *Imp √*

Other important notes:

- The critical dose is **150mg/kg in 24 hours**

  (Approximately for adults **24 tablets** = **12 grams**).

- If presents **> 8 hrs** after ingestion of a significant dose of paracetamol

  → Commence N-acetylcysteine (NAC) infusion.

- **Oral activated charcoal** is given **1g/kg (Max: 50 g) if the patient presents within 1 hour after ingesting ≥ 150mg/kg paracetamol**.

When to refer a patient with paracetamol overdose for Liver Transplantation?

	King's College Hospital criteria for liver transplantation (Paracetamol Liver Failure): ◙ **Arterial pH < 7.3**, **24 hours after ingestion** ◙ **Or all** of the following: • Prothrombin time (PT) > 100 seconds • Creatinine > 300 µmol/l • Grade III or IV encephalopathy

Key 151	**Intermittent diarrhea + weight loss (absorption problem) + Pallor (anemia) + aphthous ulcer + Hx of DM 1** Think→ **Celiac Disease**. (DM 1 and Celiac are both autoimmune diseases). Initial test → **serum tissue transglutaminase IgA (tTg-IgA)**.
Key 152	**Chronic Dysphagia + weight loss (despite age)** → **Endoscopy**

Key 153	**The most definitive (gold standard) investigation that leads to a diagnosis of inflammatory bowel disease (CD, UC) is** → **Colonoscopy.** √ Note that **fecal calprotectin** is useful as it would be raised during active colon inflammation (contrarily, it would be normal in irritable bowel syndrome). However, colonoscopy is the diagnostic approach as it would visualize the colon for the inflammation and ulcers. √ Note that **proctoscopy** is benifical for internal haemorrhoids but would not be that helpful in inflammatory bowel diseases as it would not reach and visualize the entire colon. **Important:** Note that if the question describes a case of a young patient who has features of CD or UC "young, bloody diarrhea" and the question is asking about **the most appropriate Next step**, the answer would be → **Fecal calprotectin**. However, if old, and cancer is suspected → **colonoscopy**.
Key 154	**The commonest site of fistula in Crohns's disease is the**

	→ **rectum. (Recto-vginal fistula).** **What if the rectum is not among the options?** → **ileum (ileo-vaginal fistula).**
Key 155	• Sometimes, the features of inflammatory boweel disease [IBD](eg, ulcerative colitis and crohn's) can be similar to that of irreitable bowel syndrome (IBS). **Example** (Young age, with diffuse - generalised abdominal pain and diarrhea OR constipation OR both that occur recurrently and relieved by defecation). The next most appropriate investigation would be → Fecal calprotectin (which will be raised in IBD and normal in IBS). • Although the goldstandard diagnostic modality of IBD is colonoscopy, it is invasive, costly and time-consuming. So, if the fecal calprotectin is found to be high or the patient has prominent features of IBD (eg, new onset of bloody diarrhea), we would go for colonoscopy. • Do not forget that the most appropriate investigation for suspected colon cancer (eg, old age > 50 YO with new changes in bowel habits, low Hb, weight loss, Family Hx of colon cancer, Rectal bleeding) → colonoscopy.
Key 156	Epigastric pain, vomiting, vomiting of bright red blood, Hx of taking naproxen and prednisolone for rheumatoid arthritis.

	• **Think** → Peptic ulcer. • **What is the PRIMARY diagnostic investigation**? → Gastrointestinal endoscopy. • Prolonged use of NSAIDs eg, naproxen and steroid can cause gastric elcers. • Erect chest x-ray can be useful as it would show air underdiaphragm in papteints with perforated peptic ulcer. • However, the **parimary investiagtion** that is diagnostic and therapeutic is **upper GI endocopy** as it can locate the source of bleeding and stop it).
Key 157	Old age + Hx of Smoking + Dysphagia (eg, progressive difficulty in swallowing solids) + weight loss Suspect → [Oesophageal carcinoma].
Key 158	**Artificial Nutrition** Short or long-term intervention that is used to ensure adequate nutrition and reduce the risk of aspiration in patients who are unable to swallow safely. **First: Enteral Feeding (NGT VS PEG)** **NGT = Nasogastric tube** **PEG = Percutaneous Endoscopic Gastrostomy.**

◙ Short-Term feeding

→ **NGT** "Nasogastric Tube".

- Usually used first, unless if long-term feeding is required (see below).

- **Example**: a few days after stroke (recent stroke), and the patient has started to get swallowing difficulties. We start with NGT feeding as his swallowing might improve with time. So, we feed him via NGT slowly and refer him to SALT (Speech and Language Therapist) who will assess and encourage his swallowing. If no improvement after a few weeks (Max.: 4 weeks) → PEG "Percutaneous Endoscopic Gastrostomy will be needed.

◙ Long-Term feeding

→ **PEG** "Percutaneous Endoscopic Gastrostomy feeding tube".

- It is surgery to insert a flexible tube through the abdomen into the stomach. Thus, the patient has to be fit for sedation and surgery.

- **Example (1):** A patient with an old stroke (months) and no improvement of dysphagia or swallowing, and he becomes thin (losing weight). This patient needs a long-term feeding method (PEG).

- **Example (2)**: A patient with motor neuron disease (MND) with "progressive" difficulty of swallowing. We know that MND is a chronic degenerative progressive disease. So, we do not expect improvement,

but deterioration. Therefore, a long-term feeding would be required (i.e., PEG).

The examples are important and were asked in previous exams.

Important Medical Ethics Points:

√ The next of kin (e.g., wife, brother, parents) do **not** have the legal authority to decide even if the patient lacks mental capacity.

√ If the patient who is now lacking mental capacity has an advance directive that states that he does not want to receive a specific intervention such as artificial feeding, doctors **should follow** his advance directive.

◙ Advance Directive = a living well

A legal document in which a patient writes the treatments/ the procedures that he/she does not want to receive if they become unable to make decisions.

Second: Total Parenteral Nutrition (TPN)

Total parenteral nutrition (TPN) is when the IV administered nutrition is the only source of nutrition the patient is receiving. Total parenteral nutrition is indicated when there is impaired gastrointestinal function and contraindications to enteral nutrition.

	Example: A patient is booked for total gastrectomy due to non-metastatic gastric cancer. He is in hospital as his surgery will be done in a few days. He vomits every single meal and cannot tolerate feeding due to gastric cancer (outlet-block). He is malnourished and has lost 18 Kg in the last month. What is the most appropriate feeding method before the surgery? • **The best pre-operative feeding for him** → **Total parenteral nutrition (TPN)** "A temporary method until surgery". NGT and PEG deliver nutrition to his stomach. Thus, they are not suitable as he has gastric outlet obstruction. Any nutrition directed to his stomach would not pass down to small intestine. • **The post-operative method for him would be** → **Jejunostomy feeding tube (J-tube)**. This is a plastic tube that would be inserted (during the gastrectomy surgery) through the skin of the abdomen into the jejunum so that that patient would be able to have enteral feeding after surgery. He would not have a stomach and thus he would need a feeding tube directly to his small intestine (jejunum).
Key 159	A young female presents complaining of chronic diarrhea for 6 months (up to 10 episodes of diarrhea a day). She also has recurrent anal fissures. There

	is mild generalised abdominal pain and tenderness. What is the most appropriate investigation? • It is a usual practice to **initially** perform stool culture and microscopy for any one with chronic diarrhea. • However, the **most appropriate investigation** for **Crohn's** disease (eg, chronic diarrhea, anal fissures/ fistulae, abdominal pain) is → Colonoscopy. During colonoscopy, the doctor will assess the gross features of Crohn's and will take biopsies of the affected colonic segments to look for microscopic evidence of Crohn's disease: Skip lesions, Transmural (deep Ulcers), Cobblestone appearance, Granuloma, ↑ Goblet cells.
Key 160	A young man presents complaining of recurrent (on and off) episodes of bloody diarrhea for the past 2 months, weight loss, and generalised abdominal pain and tenderness. During colonoscopy, a biopsy from the rectum was taken and revealed granulomatous inflammation. What is the most likely diagnosis? Although (chronic bloody diarrhea) is more common in ulcerative colitis than in Crohn's disease. This case is most likely a case of Crohn's disease because of the following: √ **Weight loss** is more prominent in **Crohn's** disease. √ **Granulomatous** inflammation is typically seen in **Crohn's** disease (with ↑ goblet cells). And the histology is the main method to establish diagnosis.

	On the other hand, histology of ulcerative colitis would reveal Crypt abscesses, and ↓ Goblet Cells. So, the answer is → Crohn's disease.
Key 161	**Choledolithiasis (Common Bile Duct Stones):** Where a patient presents with: √ Right upper quadrant pain. √ Jaundice (with high bilirubin). √ High ALP (Alkaline Phosphatase) ± High ALT/AST. **For Diagnosis:** • First-line investigation → **Ultrasound**. • It U/S fails to show CBD stones → MRCP. • If both U/S and MRCP did not show CBD stones → ERCP. Although ERCP is both diagnostic and therapeuric, it is the last investigating option as it is invasive. It invloves passing a flexible tube through the mouth to the stomach and duodenum. After confirming the diagnosis of bile duct stones → treat with ERCP or laparoscopic cholecystectomy with bile duct exploration. CBD = Common bile duct. MRCP = Magnetic resonance cholangiopancreatography. ERCP = Endoscopic retrograde cholangiopancreatography.

	For Management: If U/S shows CBD stones → ERCP could be used as therapeutic, or in some cases a laparoscopic cholecystectomy with bile duct exploration.
Key 162	In patients with **Oesophageal cancer with liver metastasis** → No surgery. (End-stage oesophageal cancer is inoperable). **So, how to relieve the symptom of Severe Dysphagia?** → **Oesophageal stenting √** Insert a stent into the oesophagus, it will expand and open the obstruction (Fast and Effective).
Key 163	**Quick Important GIT Notes on Previous Topics:** ◙ History of recent intake of flucloxacillin, or amoxicllin or co-amoxiclav + presenting with pruritis, fatigue, ↑ bilirubin, ↑gamma-glutamyl transferase, ↑ ALP √ Think → Cholestasis (drug-induced cholestasis).

◙ History ot taking NSAIDs (eg, ibuprofen) for a long time + presenting with vomiting of blood (ie, upper GIT bleeding due to peptic ulcer caused by the prolonged use of NSAIDs) + decreasing blood pressure, elevated heart rate.

√ **Next step** → Administration of normal saline (IV fluids). The patient shows features of significant blood loss (↓ BP, ↑HR) → Initial resuscitation with IV fluids ± blood products (if necessary) is crucial. This is to stabilise the patient haemodynamically first.

√ **What about proton pump inhibitors**? → PPIs are important in managing upper GI bleeding as it reduces acid secretion and therefore reduces bleeding. However, the priority is to stabilise the patient first.

√ **What about urgent endoscopy**? → Endoscopy should be offered to patients with acute severe upper GI bleeding who are unstable "however, stabilising the patient comes first".

√ **What about terlipressin**? → It is used for variceal bleeding, not typically for peptic ulcer bleeding.

◙ **Sudden dysphagia with uncertain diagnosis** "ie, nothing in the history points towards a specific diagnosis"

√ **Example**: Sudden onset of dysphagia and drooling while having dinner with no previous similar history, no body weight, no changes in bowel habits, no respiratory distress signs, no physical examination abnormalities.

√ **Do** → Oesophagogastroduodenoscopy (ie, upper GI endoscopy).

◙ A few days (or weeks) following the ingestion of a local street food in a different country known for hepatitis (eg, Southeast Asia), presenting with sudden onset of malaise, vomiting, yellowish sclera, which is the most likely type of hepatitis? and immunoglobulins antibodies?

→ Hepatitis A, Immunogloubulin M antibodies.

√ IgM indicates → A recent or acute infection.

√ Hepatitis (A) → Primarily spread by faecal-oral route, typically from contaminated food or water.

◙ **Q) Why there is malabsorption in celiac disease patients (what is the pathophysiological reason for steatorrhea, anemia in celiac disease)?**

→ Villous atrophy in the small intestine

(ie, Decreased surface area for absorption).

◙ Norovirus is prevalent in care home settings due to its highly contagious nature. It can cause **diarrhea** (mainly loose watery diarrhea), fatigue, low-grade fever, abdominal pain "mostly mild".

◙ **Isolated ↑ in bilirubin, predominantly unconjugated (ie, other liver function tests are within normal) + healthy patient**

	→ Gilbet's syndrome. √ Even if the stem mentions that the patient consumes alcohol, isolated ↑ in unconjugated bilirubin in a healthy patient → think Gilbert's. √ Alcohol-related liver damage has ↑ in ALT, gamma glutamyl transferase.
Key 164	**Important Note on Celiac Disease:** **If the serum tissue transglutaminase antibodies are negative but the clinical presentation is still suggestive of celiac disease (eg, diarrhea, intermittent abdominal ache especially after consuming gluten diet eg, wheat) and in the presence of serum IgA deficiency)** **→ Perform serum tissue transglutaminase antibodies using an IgG-based essay.** **After that, arrange for jejunal/ duodenal biopsy to confirm the diagnosis.**
Key 165	**Severe Flare-Up of Inflammatory Bowel Disease (IBD)** *Overview:* • **Condition:** Acute exacerbation of IBD, which includes Crohn's disease and ulcerative colitis. These are chronic inflammatory conditions of the gastrointestinal tract. *Symptoms:* • Severe abdominal pain. • Bloody diarrhea.

- Fever and malaise.
- Rapid heart rate (tachycardia).
- Weight loss and fatigue.
- Elevated inflammatory markers (e.g., CRP).
- Low hemoglobin (anemia).

Laboratory Findings:

- **Hemoglobin (↓):** Often decreased due to bleeding and inflammation.
- **White Cell Count (↑):** Elevated, indicating inflammation or infection.
- **C-Reactive Protein (↑ CRP):** Elevated, reflecting active inflammation.

Management:

1. **Initial Treatment:**
   - **Intravenous Corticosteroids:** To rapidly reduce inflammation (e.g., hydrocortisone).
2. **Supportive Care:**
   - **Hydration and Electrolytes:** IV fluids to maintain fluid and electrolyte balance.
   - **Pain Management:** Analgesics as needed.
3. **Monitoring:**
   - Frequent assessment of vital signs and symptoms.
   - Regular blood tests to monitor inflammatory markers and hemoglobin levels.
4. **Nutritional Support:**

- Bowel rest, if necessary, possibly including total parenteral nutrition (TPN) for severe cases.

Additional Considerations:

- **Broad-Spectrum Antibiotics:** If there is suspicion of an infection.
- **Iron Supplementation:** If anemia is severe.
- **Surgical Consultation:** For complications such as abscesses, fistulas, or perforation.
- **Long-Term Management:** Post-flare, maintenance therapy adjustments (e.g., immunomodulators or biologics) to prevent future flares.

Complications:

- Risk of toxic megacolon.
- Bowel perforation.
- Severe bleeding.
- Increased risk of colorectal cancer over time.

Conclusion:

A severe flare-up of IBD requires prompt medical intervention to manage acute symptoms and prevent serious complications. Initial treatment typically involves intravenous corticosteroids, supportive care, and close monitoring. Long-term management is crucial for maintaining remission and improving the quality of life for patients with IBD.

Example Scenario:

A 25-year-old female presents to the Emergency Department with a four-day history of worsening abdominal pain and frequent bloody diarrhea. She reports having more than ten bowel movements per day. She has a known history of ulcerative colitis, diagnosed three years ago. Her previous flares were managed with oral mesalazine and occasional courses of oral corticosteroids. On examination, her temperature is 38.5°C, heart rate is 120 beats per minute, and her abdominal examination reveals diffuse tenderness without guarding. Laboratory investigations reveal a raised C-reactive protein and a low hemoglobin level.

Hemoglobin: 82 g/L (115-160) White cell count: 20 x 10^9/L (4-11) CRP: 150 mg/L (<10).

Which of the following is the most appropriate initial management for this patient?

A. Iron transfusion.
B. Oral mesalazine.
C. Intravenous hydrocortisone.
D. Broad-spectrum antibiotics.
E. Intravenous cyclosporine.

Answer → C. Intravenous hydrocortisone.

Explanation

This patient presents with a severe flare of ulcerative colitis, as indicated by her history and symptoms of abdominal pain, bloody diarrhea, fever,

elevated CRP, and low hemoglobin level. Given her elevated heart rate and high white cell count, she is experiencing a severe flare.

Option Analysis:

- **A. Iron transfusion:** This might be needed for long-term management of anemia, but it is not the initial treatment for an acute flare.
- **B. Oral mesalazine:** This is typically used for maintenance therapy and is not sufficient for a severe flare.
- **C. Intravenous hydrocortisone:** Intravenous corticosteroids are the first-line treatment for severe acute flares of inflammatory bowel disease, helping to rapidly reduce inflammation.
- **D. Broad-spectrum antibiotics:** These may be used if there is suspicion of infection, but they are not the primary treatment for an acute inflammatory flare.
- **E. Intravenous cyclosporine:** This is used in certain cases of severe inflammatory bowel disease that are refractory to steroids, but it is not typically the first-line treatment for an acute flare.

Conclusion

The most appropriate initial management for this patient with a severe flare of ulcerative colitis is intravenous hydrocortisone. This helps to rapidly control the inflammation and reduce symptoms associated with the severe flare.

Quick Comparison: Acute Flare of IBD vs. Toxic Megacolon

Acute Flare of IBD:

- **Condition:** Active inflammation in Crohn's disease or ulcerative colitis without significant colonic dilation.
- **Symptoms:** Abdominal pain, bloody diarrhea, fever, elevated CRP, anemia.
- **First-line Management:** Intravenous corticosteroids (e.g., hydrocortisone) to reduce inflammation rapidly.

Toxic Megacolon:

- **Condition:** Severe complication of IBD with acute colonic distension and systemic toxicity.
- **Symptoms:** Severe abdominal distension and pain, fever, tachycardia, leukocytosis, anemia, radiographic evidence of colonic dilatation.
- **First-line Management:** Bowel rest, IV fluids and electrolytes, broad-spectrum antibiotics, intravenous corticosteroids, and urgent surgical consultation if no improvement.

Summary:

Acute Flare of Inflammatory Bowel Disease (IBD)

◙ An acute exacerbation of Crohn's disease or ulcerative colitis characterized by increased inflammation in the gastrointestinal tract.

	◙ **Important Manifestations:** √ Severe abdominal pain. √ Bloody diarrhea. √ Fever. √ Elevated inflammatory markers (↑ CRP). √ Low hemoglobin (anemia). √ Increased heart rate (tachycardia). ◙ **First-line Management:** → Intravenous corticosteroids (e.g., hydrocortisone) to rapidly reduce inflammation. ◙ **Note**: **Oral mesalazine** is typically used for maintenance therapy and is not sufficient for a severe flare. ◙ **Note**: **Broad-spectrum antibiotics** may be used if there is suspicion of infection, but they are not the primary treatment for an acute inflammatory flare.
Key 166	**Management of Significant Upper Gastrointestinal Bleed** ◙ **Clinical Presentation:** • **Symptoms**: Black, tarry stools (melena), generalized weakness, abdominal pain, vomiting blood. • **Signs of Instability**: Hypotension, tachycardia, pallor, profuse sweating.

◙ Initial Management:

- **Stabilization**: Administer intravenous fluids and transfuse packed red blood cells as needed.

◙ Diagnostic and Therapeutic Steps:

1. Urgent Endoscopy (first step after resuscitation but remains unstable).

- **Purpose**: Diagnose and treat the bleeding source.
- **Indication**: Persistent hemodynamic instability after initial resuscitation.
- **Therapies**: Clipping, banding, injection of hemostatic agents, cauterization.

2. Angiography

- **Purpose**: Visualize blood vessels and perform embolization to control bleeding.
- **Indication**: When endoscopy fails to locate or control the bleed.

3. Laparotomy

- **Purpose**: Surgical access to control bleeding.
- **Indication**: Last resort when endoscopy and angiography are unsuccessful.

◙ Pharmacological Management:

- **Proton Pump Inhibitors (PPIs)**
  - **Role**: Important for managing gastric ulcers.
  - **Guideline**: Not the primary intervention for non-variceal upper GI bleeds before endoscopy.

	• **Terlipressin**
	  ◦ **Role**: Used for bleeding esophageal varices.
	  ◦ **Guideline**: Not indicated without confirmed varices or cirrhosis.
	
	Summary Notes on Managing Upper GIT Bleeding
	
	• For significant GI bleeds with hemodynamic instability, prioritize → resuscitation with IV fluids and packed red blood cells as needed.
	
	• After resuscitation, if remains unstable → urgent endoscopy, not laparotomy.

Key 167	**Electrolyte Disturbance in Villous Adenoma** ◙ ***Clinical Scenario*** • **Patient**: 66-year-old man. • **Symptoms**: Persistent abdominal pain, changes in bowel habits, voluminous and foul-smelling stools over 8 months. • **Diagnostic Finding**: Colonoscopy reveals a large villous adenoma in the sigmoid colon. ◙ ***Most Likely Electrolyte Disturbance in villous adenoma → Hypokalemia.*** ◦ Villous Adenomas secrete large amounts of mucus. ◦ It causes excessive potassium secretion into the intestinal lumen.

	○ This results in significant potassium loss through stool, leading to hypokalemia. ◙ ***Summary on Villous Adenoma:*** **√** Patients with large villous adenomas in the colon often experience malabsorption and electrolyte disturbances due to mucus secretion. **√** The most common electrolyte imbalance is hypokalemia, caused by excessive potassium loss in the stool.
Key 168	**An Important Scenario On A Previous GIT Topic** A 35-year-old man presents with a six-month history of intermittent diarrhea, bloating after meals, and flatulence. He reports no weight loss, rectal bleeding, or significant abdominal pain. On examination, his abdominal examination is normal, and there are no signs of malnutrition. Which of the following investigations is most appropriate to request? A. Colonoscopy B. Barium swallow study C. Faecal calprotectin D. Thyroid function tests E. Tissue transglutaminase antibody (TTG) test **Answer** → E. Tissue transglutaminase antibody (TTG) test.

	Explanation: The patient's symptoms of diarrhea, bloating, and flatulence after meals without weight loss or rectal bleeding are suggestive of celiac disease. The tissue transglutaminase antibody (TTG) test is the most appropriate initial investigation for diagnosing celiac disease, as it is highly sensitive and specific. **Why Not Faecal Calprotectin?** Faecal calprotectin is a marker of intestinal inflammation and is used primarily to differentiate between inflammatory bowel disease (IBD) and irritable bowel syndrome (IBS). The patient's presentation does not strongly suggest IBD, as there are no signs of significant inflammation, weight loss, or rectal bleeding or bloody diarrhea. Thus, faecal calprotectin is not the most appropriate initial test in this case. Celiac disease, characterized by malabsorption and gastrointestinal symptoms triggered by **gluten**, is a more probable diagnosis, making the TTG test the preferred initial investigation.
Key 169	**An Important Scenario On A Previous GIT Topic** A 30-year-old man presents with complaints of increasing fatigue and intermittent abdominal pain for the past six months. He mentions experiencing occasional bouts of diarrhea mixed with blood. On further questioning, he reveals that he was diagnosed with mild ulcerative colitis two years ago but has never taken any medication for the condition. He believes his symptoms have been manageable until recently. Physical examination shows mild pallor. Blood tests reveal a hemoglobin level of 92

g/L (115-160). Which of the following is the most appropriate next step in the management of this patient?

A) Recommend dietary modifications only.

B) Azathioprine.

C) Methotrexate.

D) Ferrous sulfate.

E) Mesalazine.

Answer → E. Mesalazine.

Explanation:

• The patient's symptoms of increasing fatigue, abdominal pain, and diarrhea with blood suggest a flare of his previously diagnosed mild ulcerative colitis. This flare has likely contributed to his anemia, as indicated by the reduced hemoglobin level. Since he has not been on any treatment for his ulcerative colitis, initiating **mesalazine** is appropriate for managing mild to moderate inflammation and preventing further flare-ups.

• **Ferrous sulfate** would also be indicated to address his iron deficiency anemia secondary to chronic blood loss. However, the primary focus should be on controlling the inflammation to prevent ongoing blood loss.

• **Azathioprine** and **methotrexate** are more commonly used for more severe or refractory cases of ulcerative colitis and are not the first-line treatment for mild to moderate cases. **Azathioprine** is typically used for maintenance therapy in moderate to severe ulcerative colitis or when mesalazine is insufficient.

• **Dietary modifications** alone would not adequately address the inflammation causing his symptoms.

• **Intravenous hydrocortisone** is typically reserved for severe flares of inflammatory bowel disease (IBD) that do not respond to first-line treatments. Severe flares are characterized by more significant symptoms such as frequent bloody stools, severe abdominal pain, and marked systemic symptoms. This patient's presentation, while indicative of a flare, does not yet reach the severity that would necessitate intravenous corticosteroids.

Summary:

The patient is experiencing a mild to moderate flare of ulcerative colitis, leading to symptoms of fatigue, abdominal pain, and bloody diarrhea, causing anemia.

• Mesalazine is the most appropriate initial treatment to control inflammation and prevent further flares.

• Ferrous sulfate may also be used to treat anemia, but the primary goal is to manage the underlying inflammatory condition.

	• Intravenous hydrocortisone is reserved for more severe flares that do not respond to first-line therapies.
Key 170	**Liver Cirrhosis (Scenario + Explanation)** A 55-year-old woman presents to the clinic with progressive abdominal distension and discomfort over the past three months. She reports increased fatigue, loss of appetite, and significant weight loss. On examination, the abdomen is tense, and a fluid wave test is positive. Additionally, multiple spider naevi are noted on her chest and upper abdomen. The serum-ascites albumin gradient (SAAG) is measured at 13 g/L. Investigations are done and are as follows: Sodium: 136 mmol/L (135-145) Potassium: 4.0 mmol/L (3.5-5) Urea: 6 mmol/L (2.0-7) Creatinine: 120 µmol/L (70-150) eGFR: 60 mL/min (>90) Bilirubin: 25 µmol/L (3-17) Alanine transferase (ALT): 105 U/L (5-35) Alkaline phosphatase (ALP): 140 U/L (30-150) Aspartate transaminase (AST): 90 U/L (5-35) Albumin: 30 g/L (35-50)

Which of the following is the most likely underlying cause of her symptoms?

A. Pancreatic cancer.

B. Cirrhosis.

C. Tuberculosis.

D. Heart failure.

E. Nephrotic syndrome.

Answer → B. Cirrhosis.

The patient presents with signs and symptoms consistent with chronic liver disease, including progressive abdominal distension (ascites), discomfort, fatigue, loss of appetite, significant weight loss, and the presence of multiple spider naevi. The positive fluid wave test and the high serum-ascites albumin gradient (SAAG) of 13 g/L indicate ascites due to portal hypertension.

Supporting Points for Cirrhosis:

◙ Clinical Symptoms:

- Progressive abdominal distension and discomfort.
- Increased fatigue and loss of appetite.
- Significant weight loss.
- Presence of multiple spider naevi, which are common in chronic liver disease.

◙ Physical Examination:

• Tense abdomen with positive fluid wave test (indicative of ascites).

• Multiple spider naevi on chest and upper abdomen.

◙ Laboratory Findings:

• Elevated Liver Enzymes (ALT, AST): indicate liver injury.

• Low Albumin: indicative of reduced liver synthetic function.

• High SAAG: is strongly suggestive of portal hypertension, a hallmark of cirrhosis.

Key Points

1. **High-Gradient Ascites (SAAG ≥ 11 g/L)**
   - Indicates ascites due to portal hypertension.
   - Common causes: Cirrhosis, heart failure.
2. **Low-Gradient Ascites (SAAG < 11 g/L)**
   - Suggests ascites due to non-portal hypertension causes.
   - Common causes: Malignancy (e.g., ovarian, pancreatic, gastric, colon cancers), tuberculosis, pancreatitis, nephrotic syndrome.

◙ Differential Diagnosis Consideration:

• **Pancreatic Cancer**: Can cause ascites but usually presents with other symptoms like severe abdominal pain and jaundice, and typically has a low SAAG.

• **Tuberculosis**: Can cause ascites but usually presents with systemic symptoms and signs of infection, and typically has a low SAAG.

• **Heart Failure**: Can cause ascites, but other signs like elevated jugular venous pressure and peripheral edema would be more prominent.

• **Nephrotic Syndrome**: Usually presents with significant proteinuria, hypoalbuminemia, and generalized edema rather than localized ascites with a high SAAG.

Given the clinical picture and the laboratory findings, **cirrhosis** is the most likely underlying cause of the patient's symptoms.

Extra Important Points: Ascites due to liver cirrhosis:

◙ **Investigation?**

→ **Ascitic fluid aspirate analysis: culture, cell count "neutrophil count".**

◙ **Management of ascites due to cirrhosis? → Spironolactone.**

◙ **Other important lines:**

√ **If high neutrophils → IV antibiotics.**

√ **If albumin is low → albumin infusion.**
